VT STORM

Jacqueline Joza

Cardiac Electrophysiologist

McGill University Health Centre

Conflict of Interest Disclosures

- **Grants/research support**: Medtronic, Heart and Stroke

Objectives

1. Understand basic mechanisms and etiologies of VT storm
2. Appreciate the approach to management
3. Discussion of clinical scenarios where invasive EP management should be considered

Background: **VT/VF Storm**

3 or more episodes of VT/VF within 24 hours requiring either ATP or cardioversion/ Defibrillation

- 1 episode is defined as sustained or unstable VT or VF
- with more than 5 minutes between episodes

Up to 3.5- 4% of the primary prevention patients will be affected

Up to 10- 40% of the secondary prevention patients will be affected

Kumar S, et al.. *J Cardiovasc Electrophysiol*. 2017;28(1):56-.

(Ventricular Arrhythmias VAs) predispose the failing heart to more pump failure

COMPANION Study: NYHA III/ambulatory IV comparing OMT to CRT: presence of an appropriate shock for sustained VT associated with

- increased risk of SCD: OR 2.97 and
- increased risk for pump failure death or hospitalization: OR 2.45
- Event rate of pump failure death or hospitalization due to HF reached near 50% at 1 year in those patients who received appropriate defibrillation

Saxon et al. Circulation.114(25); 2766-

ICD shocks are BAD

- Shocks are Painful
- Increase Anxiety and Depression
- Decrease Quality of Life
- Increase Heart Failure
- Increase Mortality

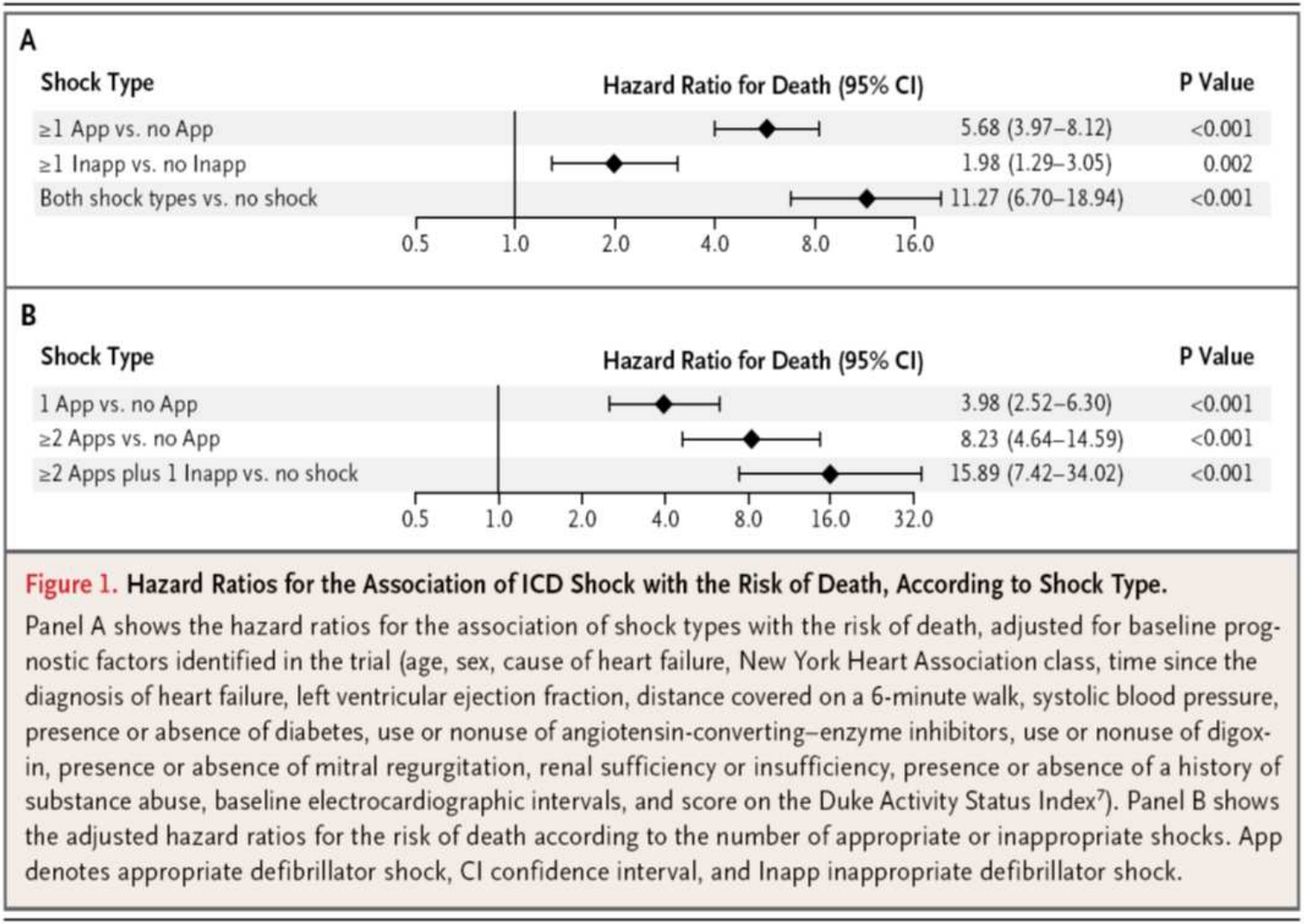


Figure 1. Hazard Ratios for the Association of ICD Shock with the Risk of Death, According to Shock Type.

Panel A shows the hazard ratios for the association of shock types with the risk of death, adjusted for baseline prognostic factors identified in the trial (age, sex, cause of heart failure, New York Heart Association class, time since the diagnosis of heart failure, left ventricular ejection fraction, distance covered on a 6-minute walk, systolic blood pressure, presence or absence of diabetes, use or nonuse of angiotensin-converting–enzyme inhibitors, use or nonuse of digoxin, presence or absence of mitral regurgitation, renal sufficiency or insufficiency, presence or absence of a history of substance abuse, baseline electrocardiographic intervals, and score on the Duke Activity Status Index[7]). Panel B shows the adjusted hazard ratios for the risk of death according to the number of appropriate or inappropriate shocks. App denotes appropriate defibrillator shock, CI confidence interval, and Inapp inappropriate defibrillator shock.

ALTITUDE survival study: very long-term follow-up of ICDs

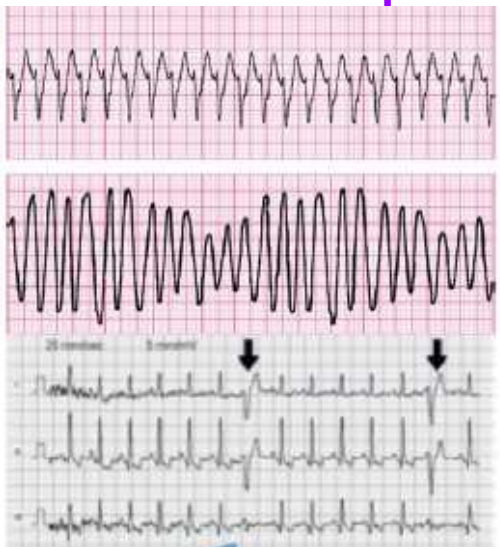
- Subsequent analysis: <u>appropriate</u> shock increases mortality
- Polymorphic worse than monomorphic

Poole; NEJM 2008; 359; Saxon et al. Circulation. 2010;122:2359–2367

Pathophysiological Cycle of Ventricular Arrhythmias and Progressive Pump Failure

Myocardial fibrosis
Progression of arrhythmogenic substrate
Electrical remodeling (connexin 43, Ca cycling, QT and QRS prolongation

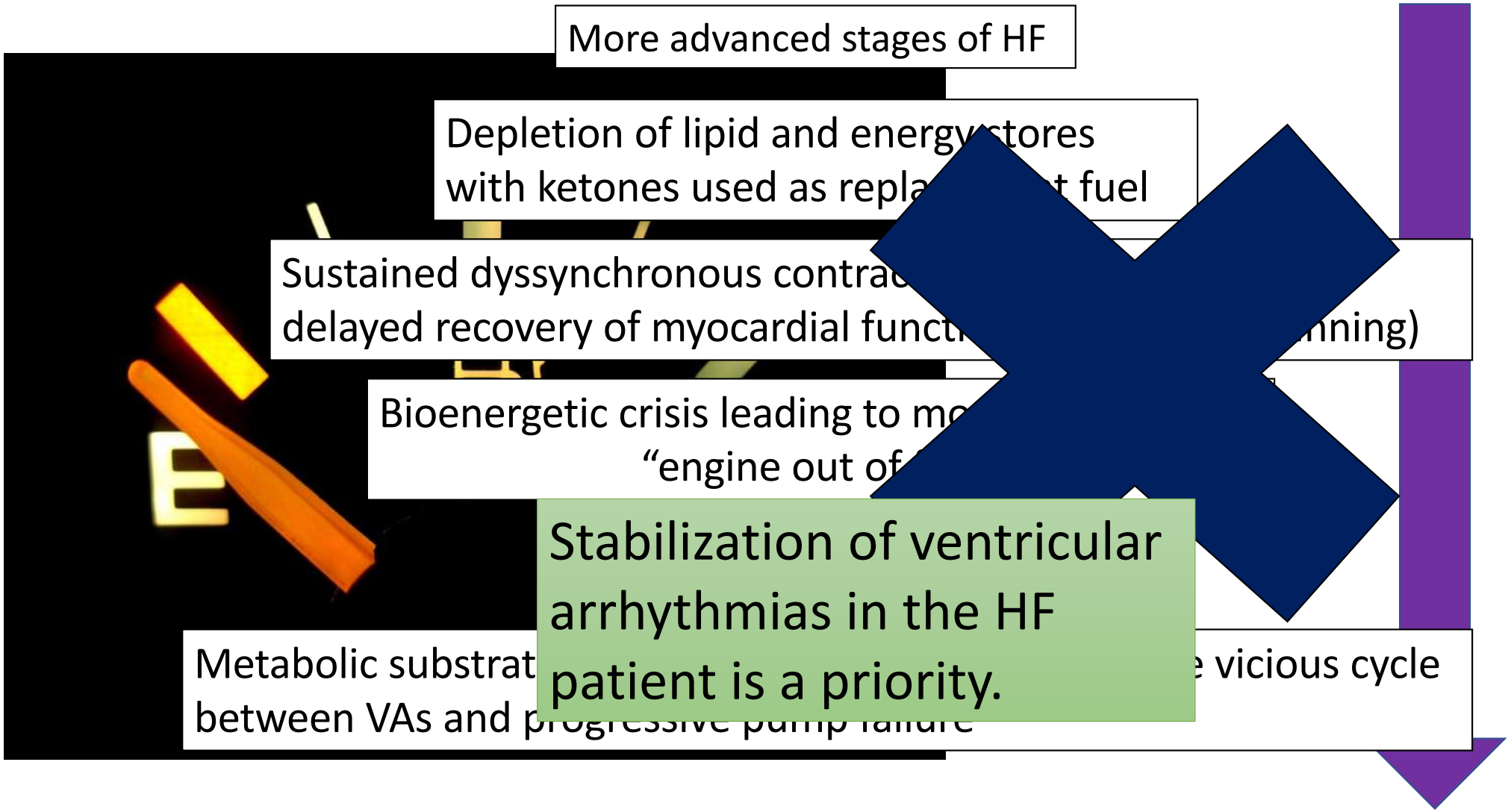
Ventricular Arrhythmias
-Frequent PVCs
-Couplets
-NSVT
-Sustained VT
-VF



Maladaptive neurohormonal and metabolic reprogramming

HF disease progression
-maladaptive hypertrophic and fibrotic myocardial remodeling
-mechanical-bioenergetic inefficiency
+- dyssynchronous contraction

Sicker Patient
Hemodynamic decompensation


More advanced stages of HF
Depletion of lipid and energy stores
with ketones used as repla t fuel
Sustained dyssynchronous contrac
delayed recovery of myocardial funct nning)
Bioenergetic crisis leading to mo
“engine out of
Stabilization of ventricular arrhythmias in the HF patient is a priority.
Metabolic substrat e vicious cycle
between VAs and progressive pump failure

Cardiac resynchronization therapy has been proven to decrease fibrosis and improve myocardial remodeling

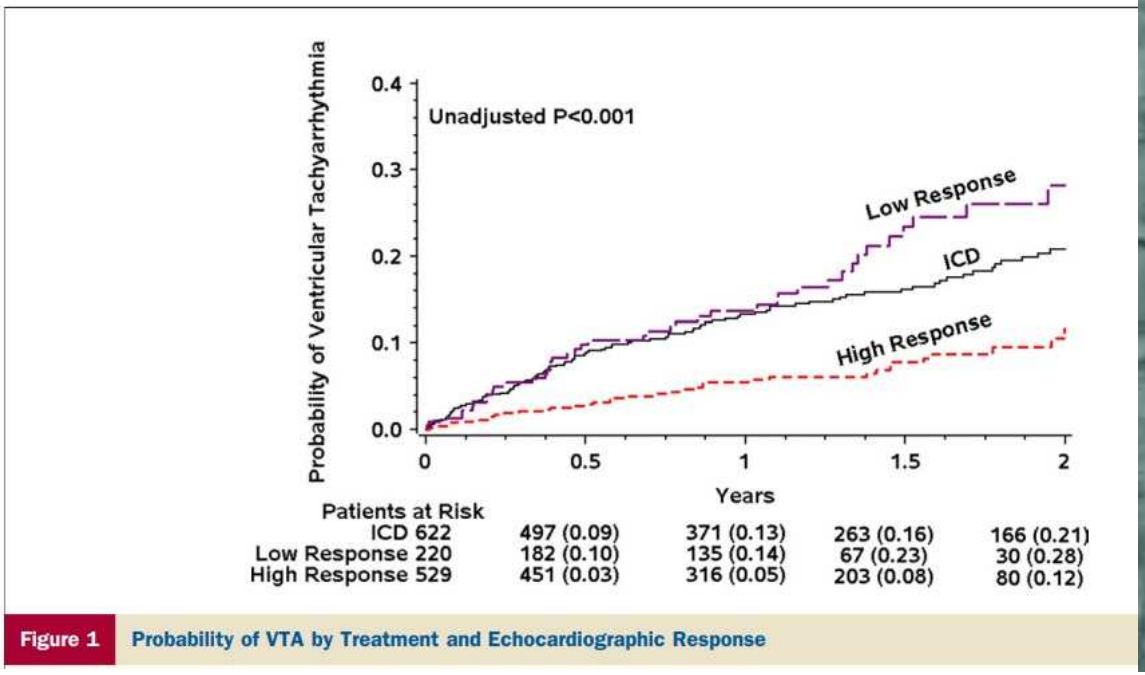
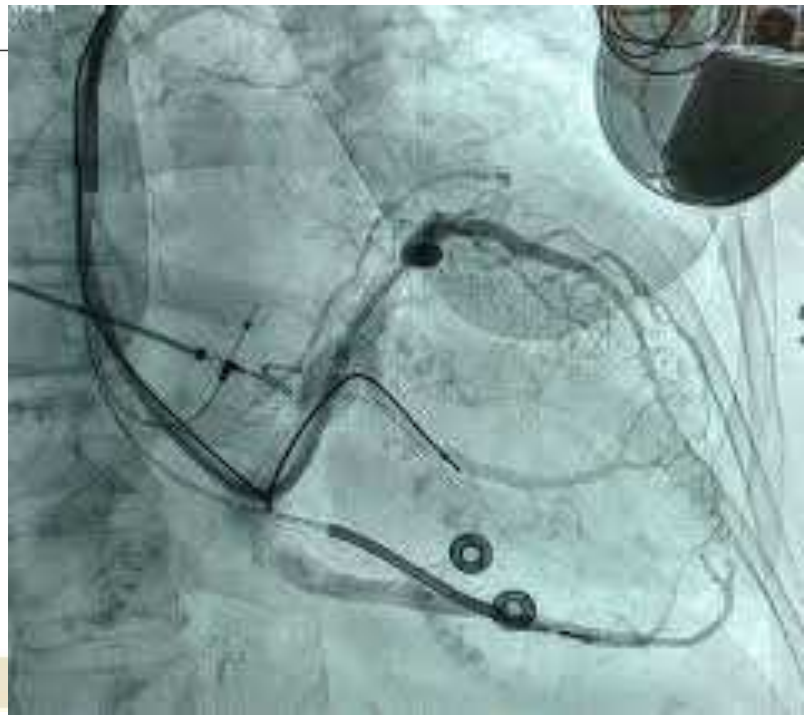


Figure 1 Probability of VTA by Treatment and Echocardiographic Response



Less sick patients: NYHA Class I and II

Barsheshet et al. JACC 2011;57:2416

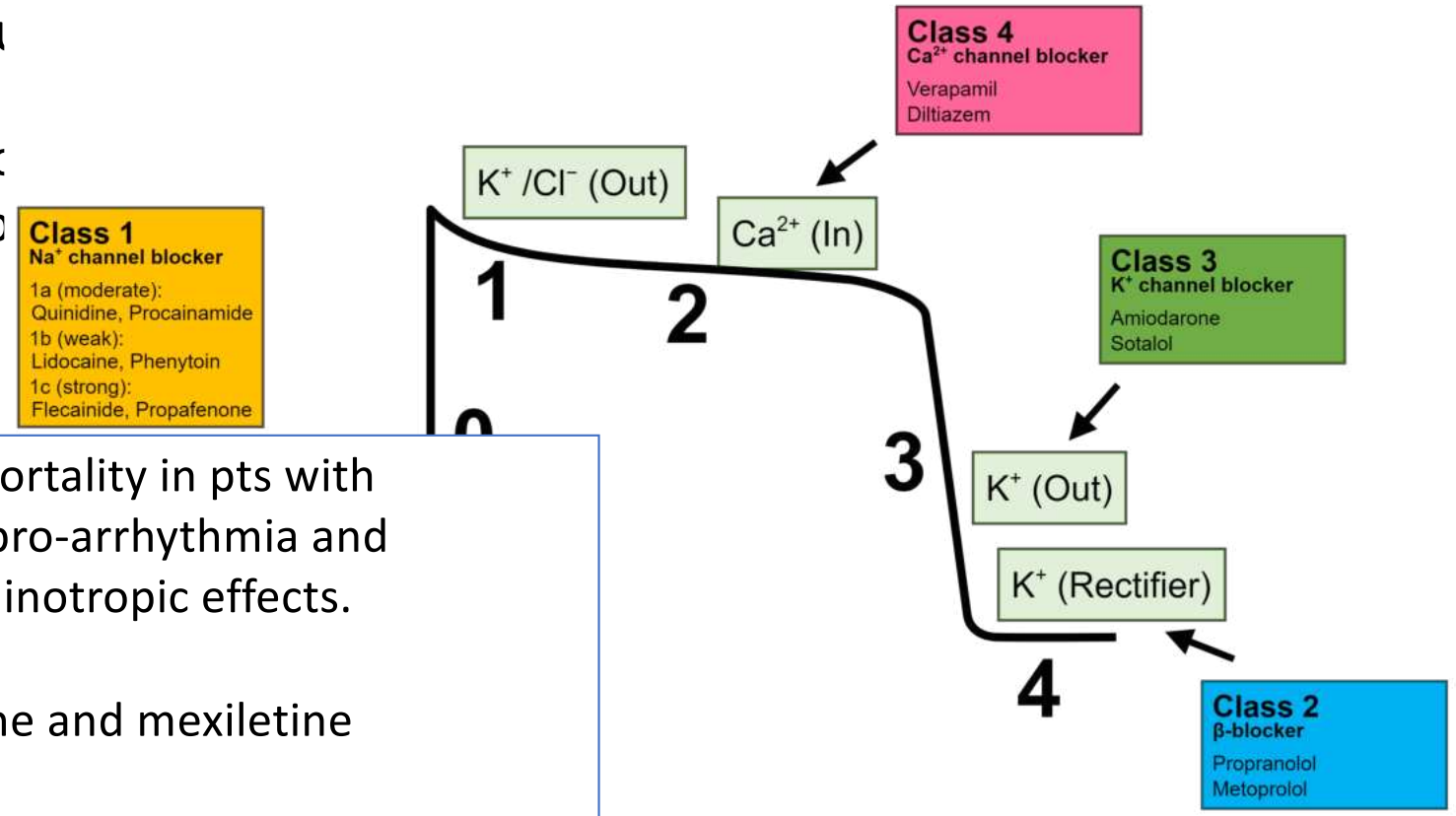
Not all mechanisms of HF disease regression and the therapeutic interventions that produce them, can reduce the burden of VAs

Still a high prevalence of VAs when HF is ‘reversed’ in LVAD patients, particularly in those with pre-operative VT

- Reversing HF and decreasing mortality from pump failure in a significant subgroup of NYHA class III and IV HF patients does NOT reduce the presence of sustained VAs of the risk of SCD

Antiarrhythmic Dru

- Very little data fo
- Negative inotrop



Class I: Increased mortality in pts with structural disease: pro-arrhythmia and significant negative inotropic effects.

Exceptions: quinidine and mexiletine

<u>Quinidine:</u> minimal inotropic properties
<u>Mexiletine:</u> can worsen hemodynamic status in severe LV dysfunction due to increase in SVR with decrease CO/SV

TABLE 2 Summary of Randomized Controlled Trials Testing AAD Therapy Versus Standard Medical Therapy for the Prevention of VAs in Patients With ICDs

	N Included		Age, yrs		ICM		Ejection Fraction		ICD Appropriate Therapy		Deaths	
First Author, Year (Ref. #)	AAD	CTRL	AAD	CTRL	AAD	CTRL	AAD	CTRL	AAD	CTRL	AAD	CTRL
Kühlkamp et al., 1999 (112)	46	47	59 ± 18	64 ± 17	31 (67)	28 (60)	35 ± 8	38 ± 19	15 (33)*	24 (51)*	4 (9)	3 (6)
Pacifico et al., 1999 (43)	151	151	63 ± 11	61 ± 11	110 (73)	100 (66)	37 ± 12	39 ± 14	33 (22)*	49 (32)*	4 (3)	7 (5)
Kettering et al., 2002 (113)	50	50	59 ± 12	60 ± 9	35 (70)	38 (76)	38 ± 15	38 ± 14	30 (60)	33 (66)	6 (12)	8 (16)
Dorian et al., 2004 (42)	419	214							7 (59)*	136 (64)*	13 (3)	7 (3)
Singer et al., 2004 (44)	135	37							NA	NA	2 (2)	3 (8)
Connolly et al., 2006 (41)												
Amiodarone	140	138							5 (11)*	45 (33)	6 (4)	2 (1)
Sotalol	134								8 (28)		4 (3)	
Kowey et al., 2011 (114)												
Celivarone	324	109	64 ± 10	65 ± 12	225 (69)	86 (79)	29 ± 8	29 ± 8	194 (59)	66 (61)	28 (9)	6 (6)
Amiodarone	53		67 ± 8		36 (68)		29 ± 8		20 (38)*		9 (17)	

Data in favour of AADs to prevent ventricular arrhythmias becomes very murky

Systematic review of these 8 RCTs: 2,300 patients followed for mean 15 months

- 34% reduction in VA episodes leading to appropriate ICD therapy
- No effect on all-cause mortality
- Only studies evaluating amiodarone showed significant benefit
- Subgroup analyses of primary prevention ICD trials + large Afib trials have suggested increased death with amio

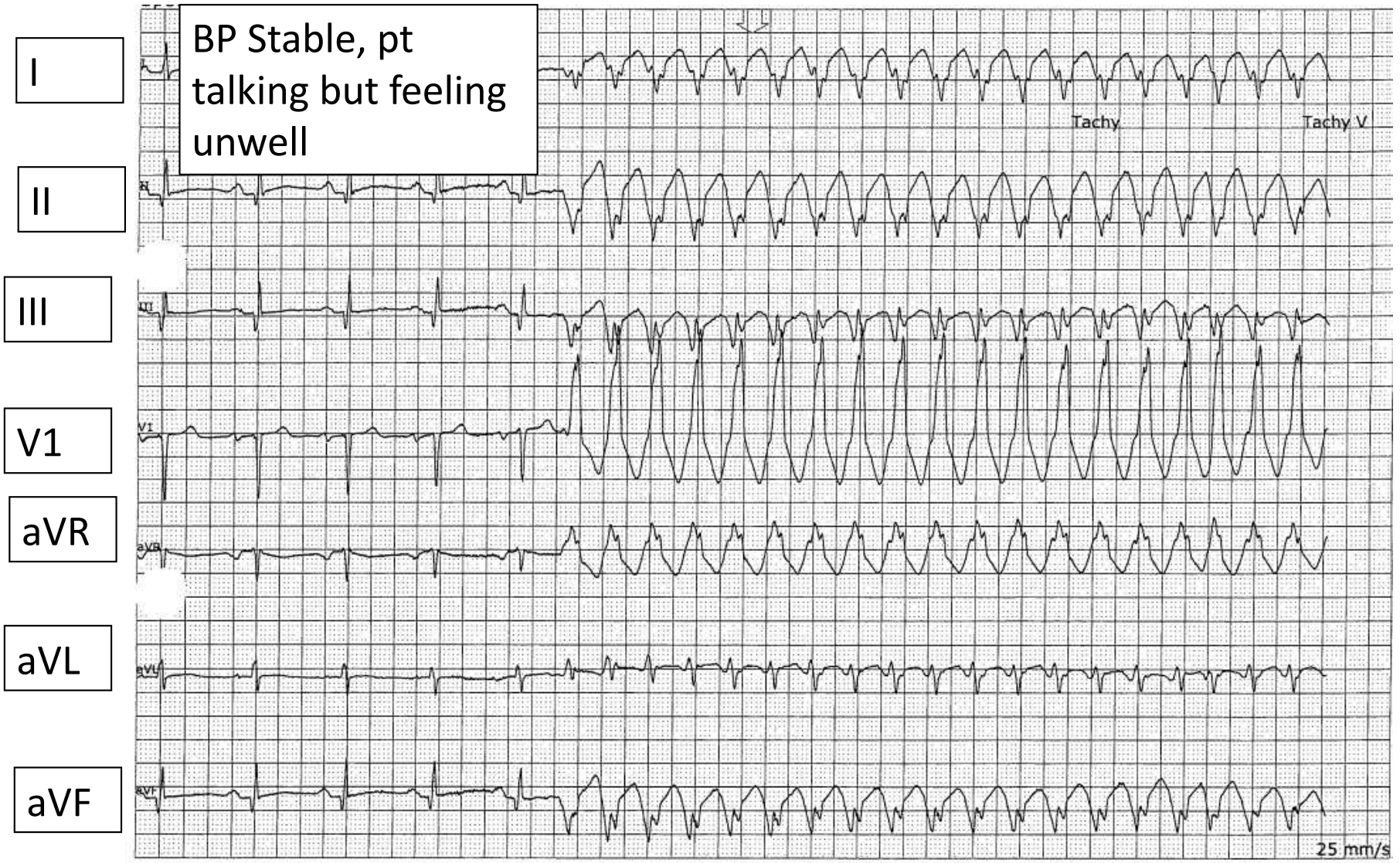
Case 1: 2017: 55 M. AB

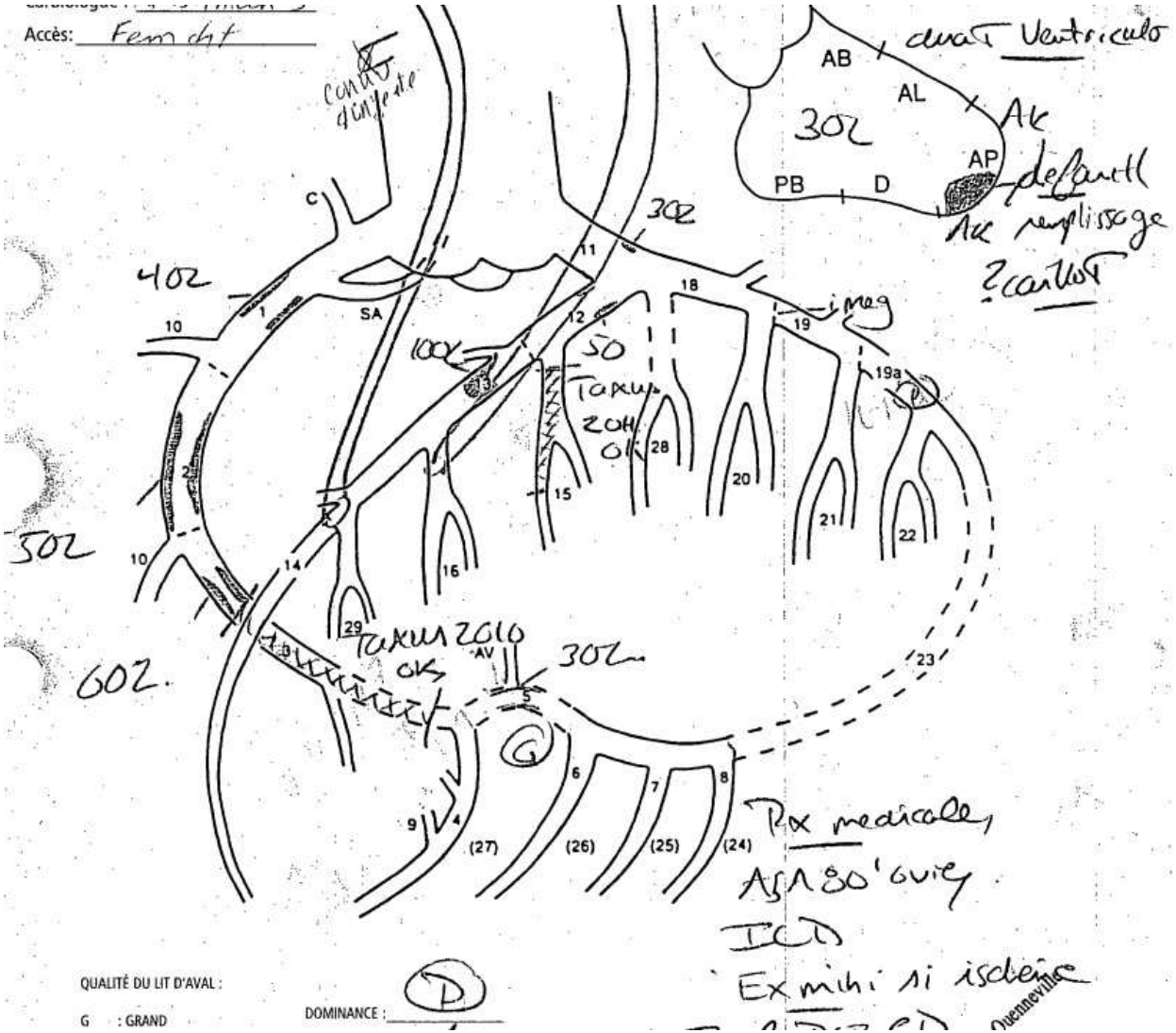
55M Construction worker.

PMHx: Hypertension, DM-2, familial hypercholesterolemia, Myocardial infarction 1994, 2010; CABG x 3 July 2010. Ex-smoker – stopped in 2006.

PCI December 2010 (2 blocked bypasses 6 months post cabg), PCI 2011 LVEF 45%

HPI: Presents with severe dyspnea and palpitations to emergency department

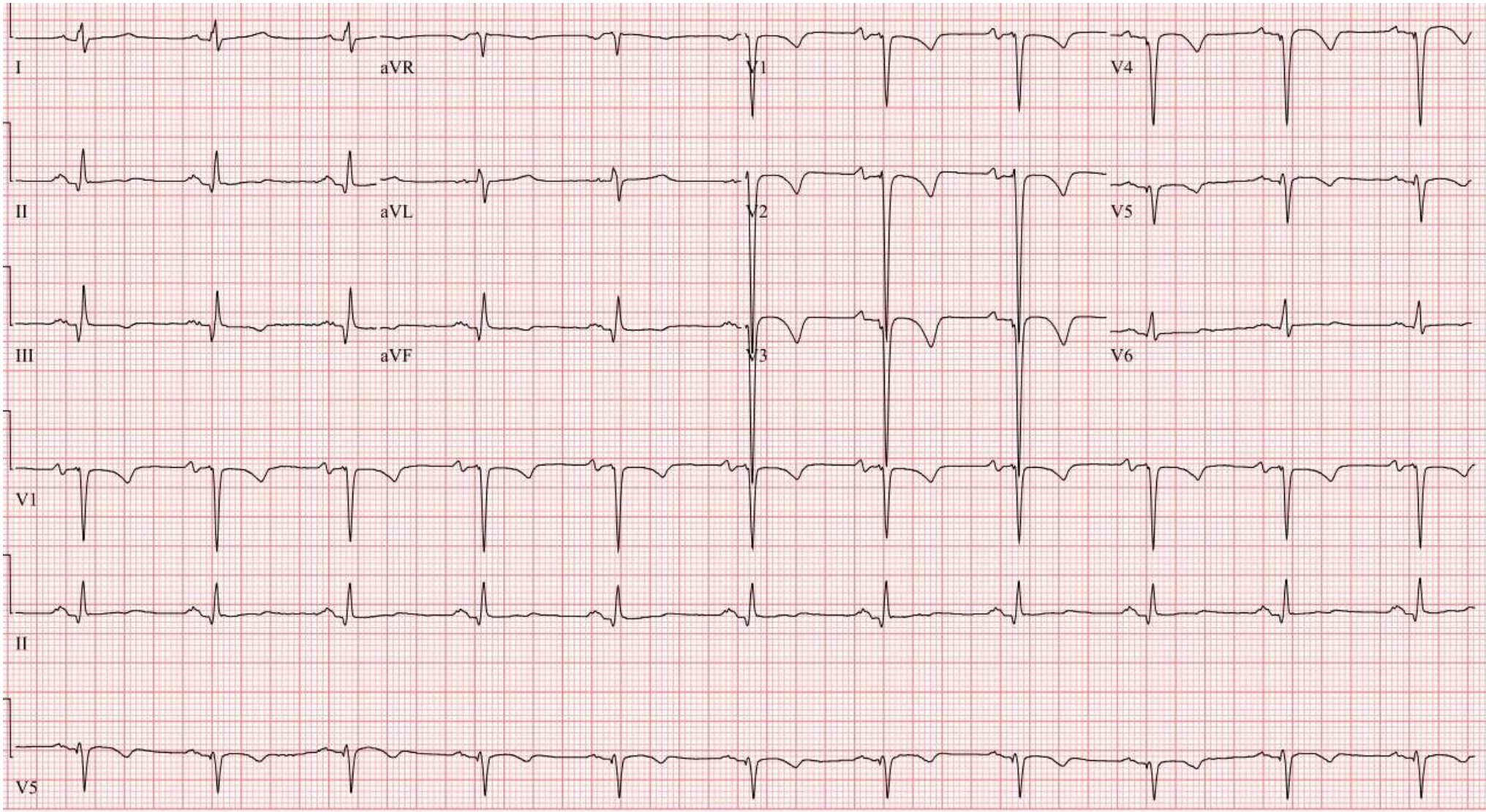

I
II
III
V1
aVR
aVL
aVF
BP Stable, pt talking but feeling unwell
Tachy
Tachy V
25 mm/s

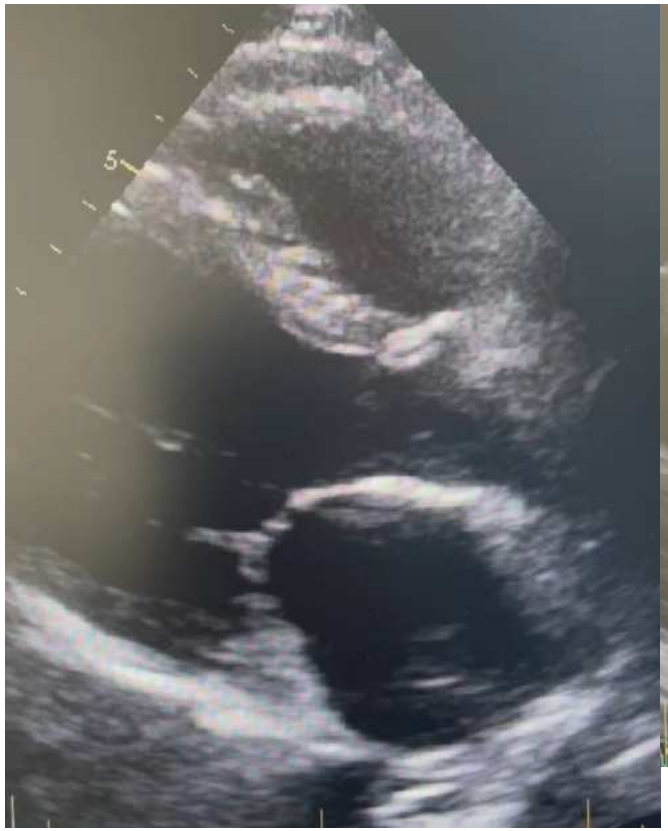
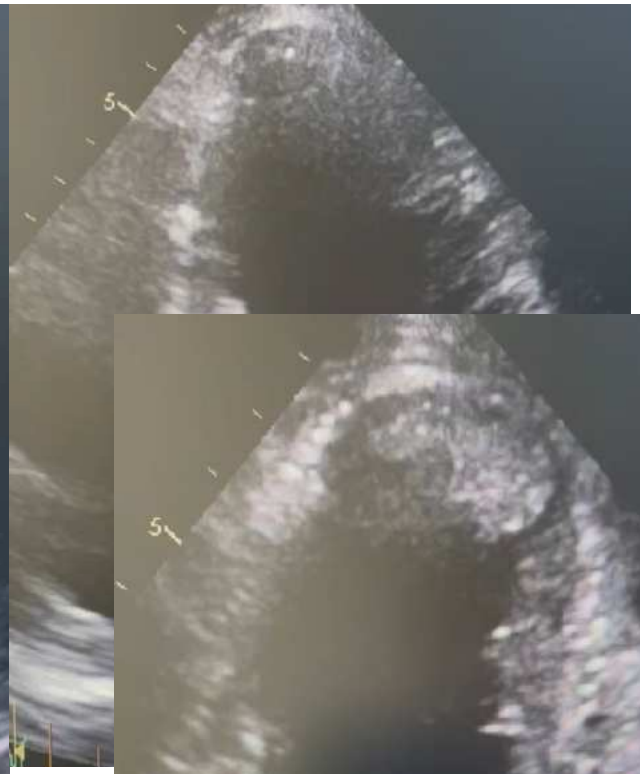
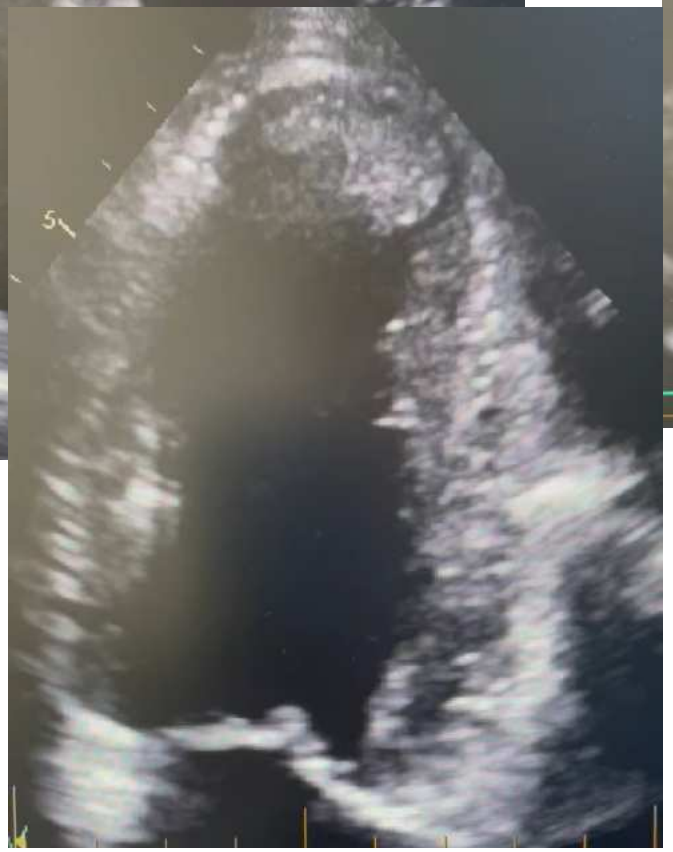
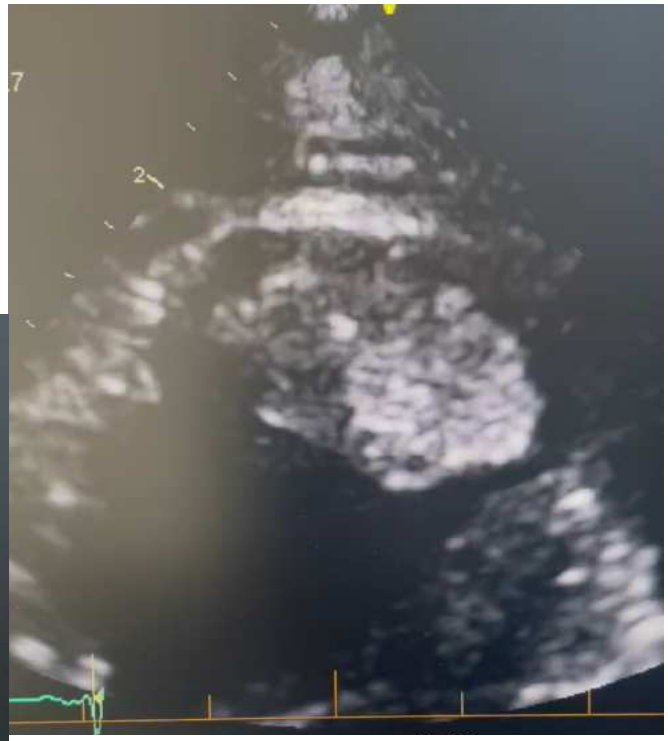


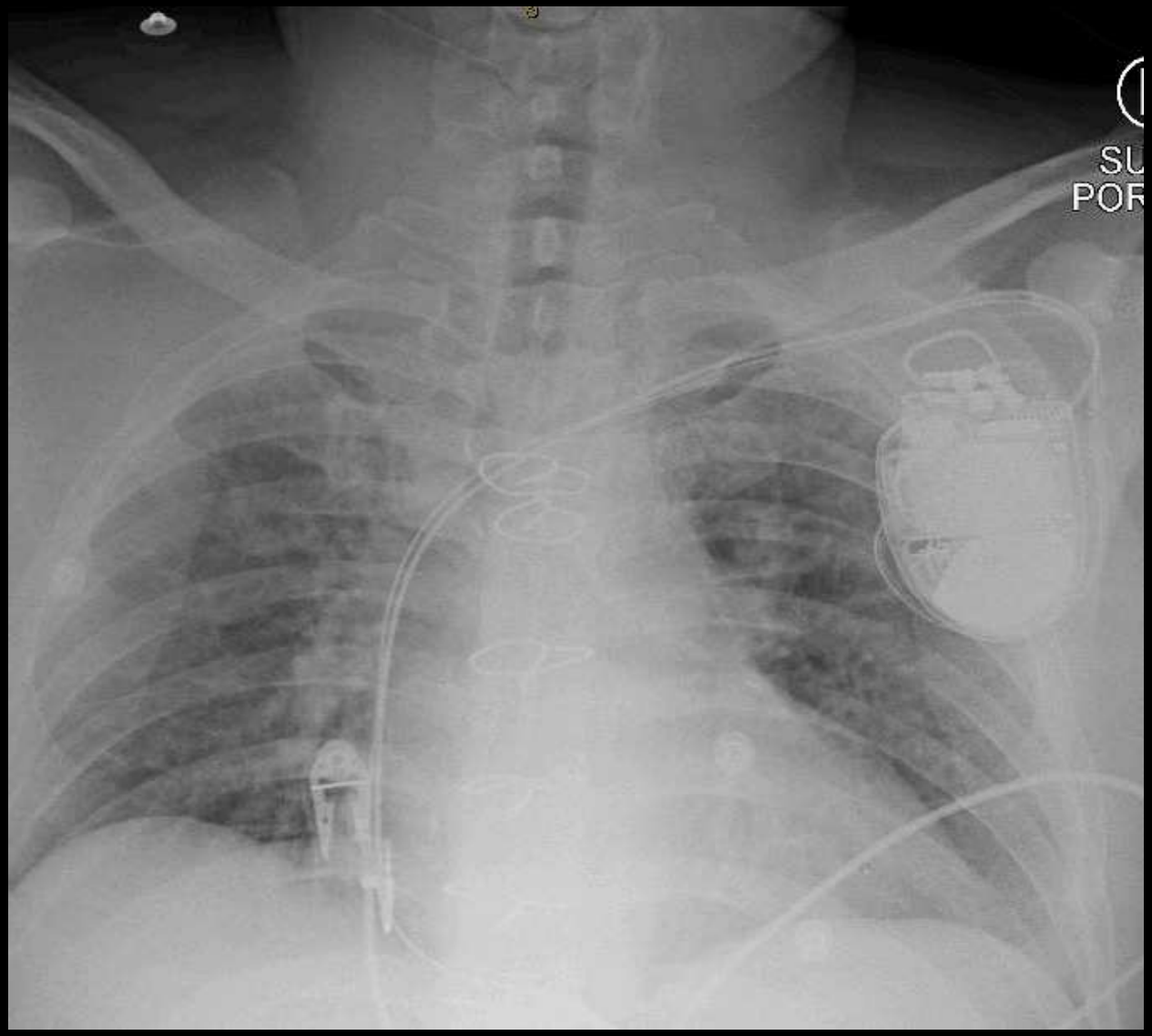
CABG:
-RIMA to LAD, but feeding very small LAD (patent)
-LIMA to Diag (patent)
-SVG to RCA (occluded)

100% mid LAD,
-prior D1 stent ok
-prior distal RCA stent ok
-40-60% RCA

-?clot in apex


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5



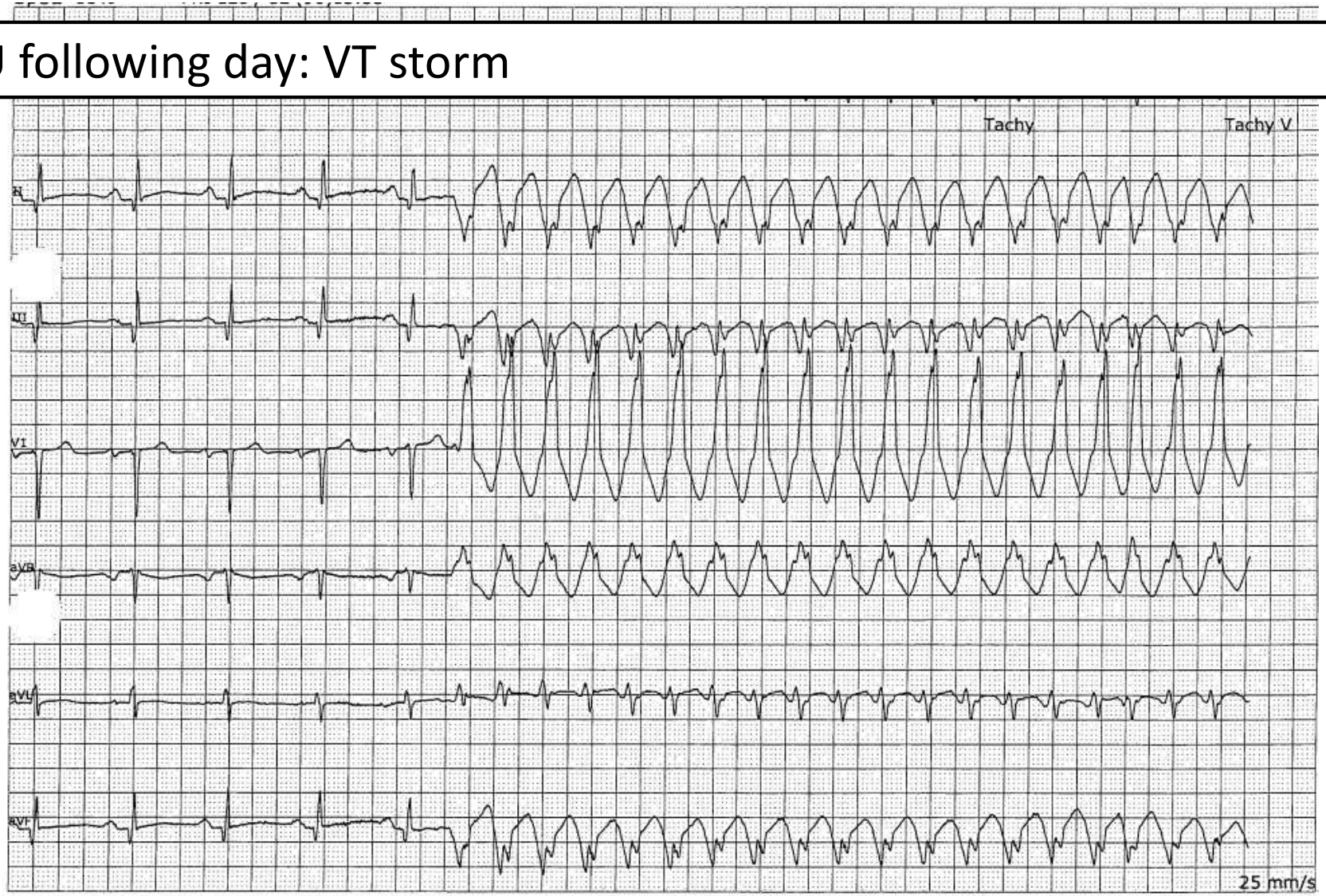
VT during ICD implant: did not terminate with ventricular overdrive pacing

-Sedated and defibrillated

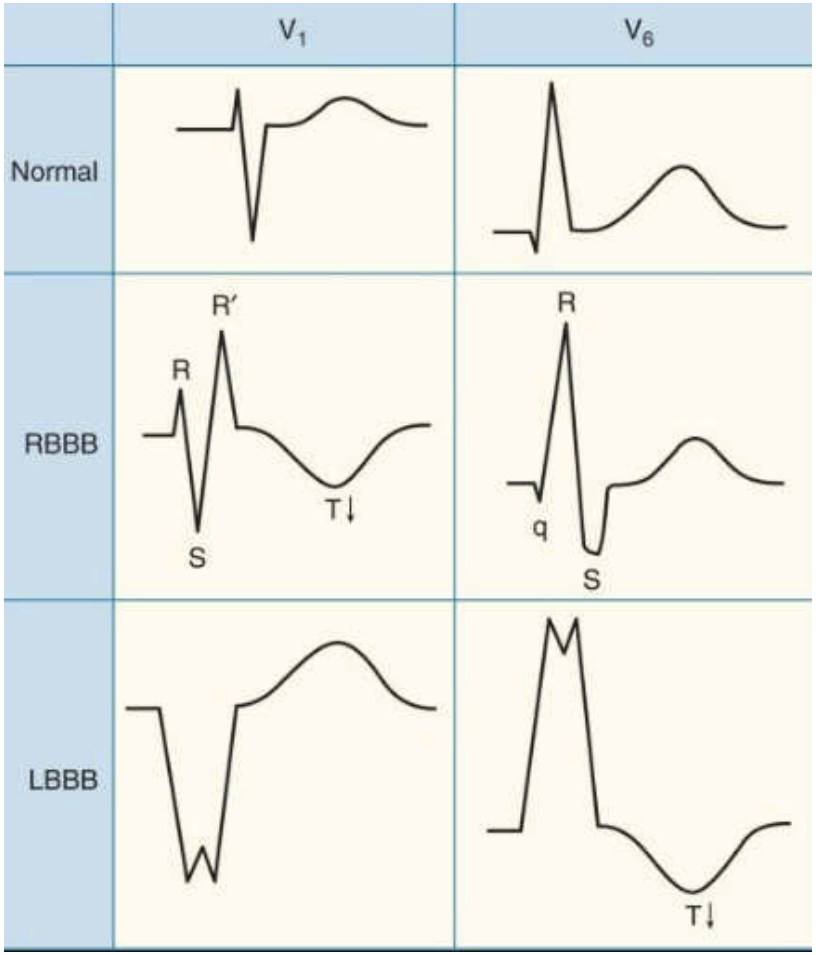
-ICD in place, patient transferred to CCU. Sotalol120mg BID started ICD set to give multiple ATPs at 133bpm, but no shock given that the VT was hemodynamically stable (avoid shock while awake)

Apixaban started that eve.

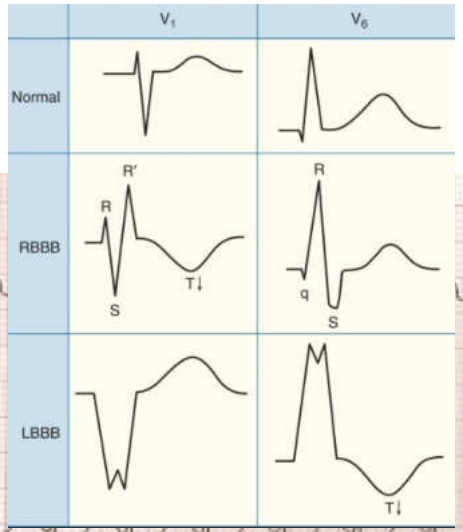
In CCU following day: VT storm



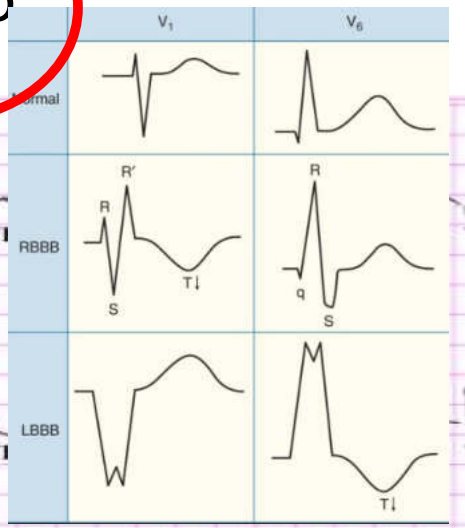
1. Evaluate the rhythm, and Stabilize hemodynamically



SVT or VT?

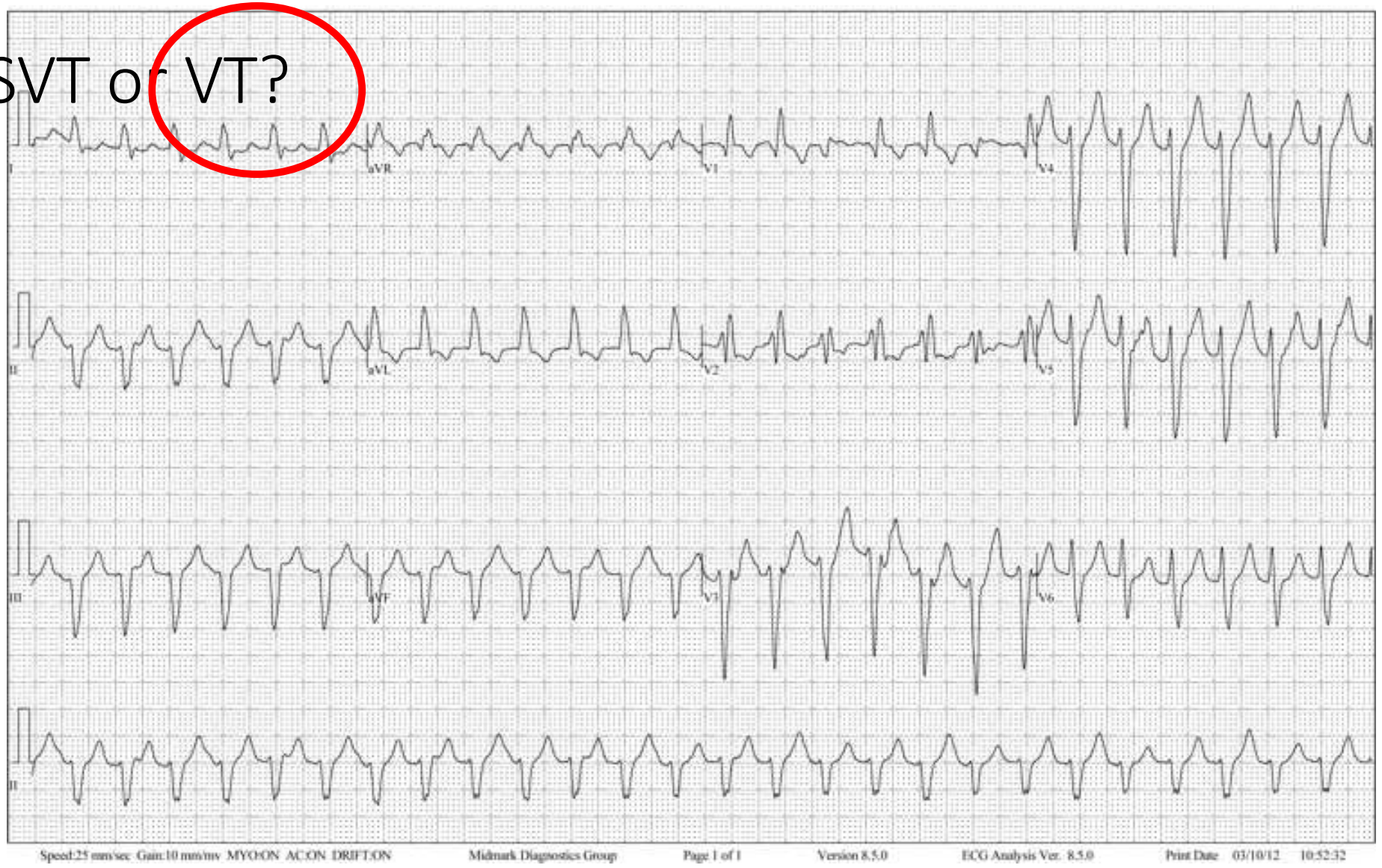


SVT or VT?


V1
V6
Normal
RBBB
R'
R
S
T↓
R
q
S
LBBB
T↓

I
aVR
II
aVL
III
aVF
V1
V2
V3
V4
V5
V6
V1

SVT or VT?


I
aVR
V1
V4
II
aVL
V2
V5
III
VF
V3
V6
II
Speed:25 mm/sec Gain:10 mm/mv MYO:ON AC:ON DRIFT:ON
Midmark Diagnostics Group
Page 1 of 1
Version 8.5.0
ECG Analysis Ver. 8.5.0
Print Date 03/10/12 10:52:32

1. Evaluate the Rhythm and Stabilize hemodynamically

2. **Evaluate for triggers.** This patient has monomorphic VT, therefore scar-related and not ischemia. Also rule out electrolyte disturbance, fever/infection, proarrhythmic drug effects..

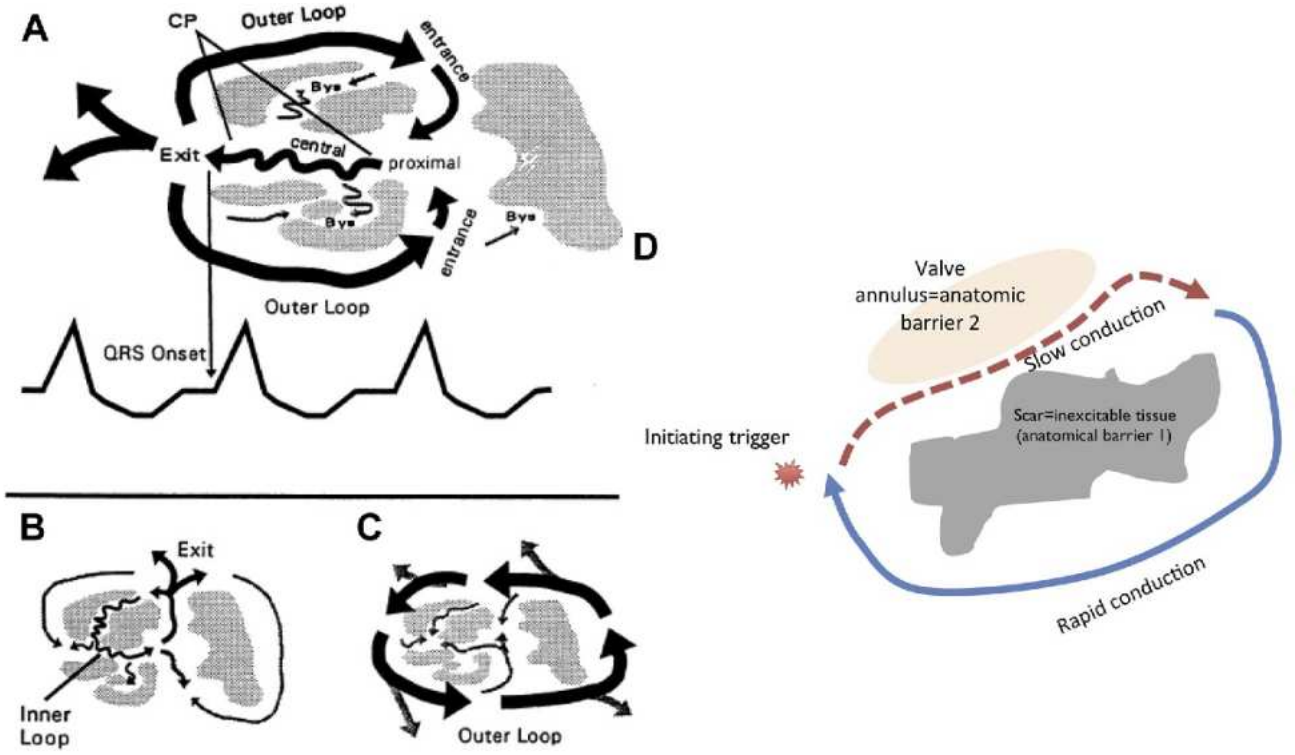


Fig. 1 Anatomic models of reentrant VT. (A–C) Three hypothetical reentrant VT circuits. Gray areas are electrically

Management of VT STorm

1. Stabilize hemodynamically
2. Evaluate for triggers. This patient has monomorphic VT, therefore scar-related.

3. Antiarrhythmic therapy:

- **Start lidocaine**
  - **Excellent for early post-myocardial infarction arrhythmias**
  - **Excellent for polymorphic VT/VF**
  - **Good for scar-related monomorphic VT, particularly if patient considered for ablation (rapid on/off pharmacokinetics) – can be stopped just before ablation to allow mapping during VT**

  - **Amiodarone: can be considered (especially in patients not on chronic therapy with amiodarone) although prefer to avoid in case an ablation procedure is planned**

European Heart Journal (2017) **38**, 1329–1335
doi:10.1093/eurheartj/ehw230

Tachycardia terminated within 40 minutes in 62% vs 28% favouring procainamide over amiodarone

Approx 14-16 min. to terminate in each arm

Fewer 'adverse effects' in procainamide arm

Wide QRS tachycardia

Randomized to:

- procainamide 10mg/kg over 20 minutes vs
- amiodarone 5mg/kg over 20 minutes

Study period: 40 minutes

Table 2 **Safety and efficacy of study drugs in the entire population**

	All patients ($n = 62$)	Procainamide ($n = 33$)	Amiodarone ($n = 29$)	*P*
Major cardiac adverse events during study period	15 (24)	3 (9)	12 (41)	0.006
Total adverse events during study period	22 (35)	8 (24)	14 (48)	0.052
Time to adverse event (min)	17 ± 9	17 ± 12	16 ± 7	0.7
Tachycardia termination during study period	33 (53)	22 (67)	11 (38)	0.026
Time to tachycardia termination (min)	14 ± 9	14 ± 10	16 ± 5	0.3
Total adverse events during the observation period	15 (24)	6 (18)	9 (31)	0.24

Values are *n* (%) and mean ± SD.

Table 4 **Cardiac adverse events during the study period (40 min)**

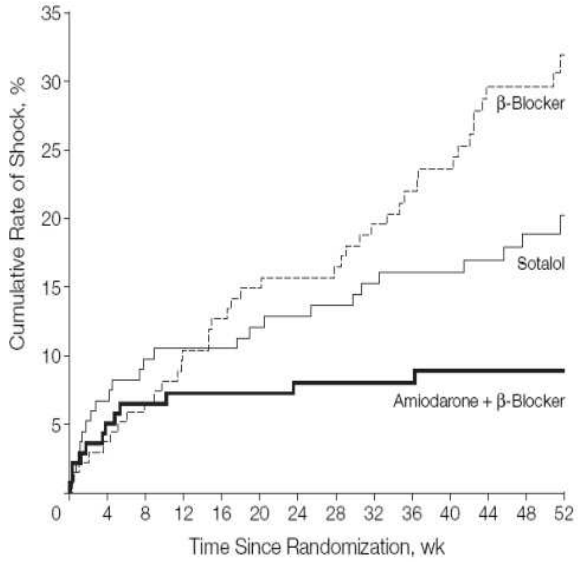
Adverse event	Procainamide	Amiodarone
Major cardiac adverse events during study period		
Acute pulmonary oedema requiring DCCV	0	2
Severe hypotension requiring cessation of infusion	1	1
Severe hypotension requiring immediate DCCV	2	6[a]
Peripheral hypoperfusion and/or dyspnoea with severe hypotension requiring immediate DCCV	0	3
Other adverse events during study period		
Hypotension		
Adverse events		
Acute pulmo		
Dyspnoea with peripheral hypoperfusion	1	0
Hypotension	3	5
Sinus bradycardia	1	0
Arrhythmic storm and cardiogenic shock	0	1
Neumothorax	0	1
Acute myocardial infarction 4 h after drug administration	0	1

Procainamide therapy had less major cardiac adverse events (HF and hypotension predominantly) and higher proportion of tachycardia termination within 40 minutes

Comparison of β-Blockers, Amiodarone Plus β-Blockers, or Sotalol for Prevention of Shocks From Implantable Cardioverter Defibrillators

The OPTIC Study: A Randomized Trial

Figure 2. Cumulative Rate of Shock for the 3 Treatment Groups by Time Since Randomization



No. at Risk					
β-Blocker	138	119	109	91	42
Sotalol	134	118	108	94	35
Amiodarone + β-Blocker	140	124	115	106	56

Log-rank $P<.001$ for amiodarone plus β-blocker vs β-blocker alone, log-rank $P=.02$ for amiodarone plus β-blocker vs sotalol alone, and log-rank $P=.055$ for sotalol vs β-blocker.

Clear benefit with amiodarone + beta blocker as compared to sotalol alone, or beta blocker alone

Classically beta 1 receptors get down-regulated in heart failure, therefore non-selective BB (propranolol, nadolol) have further advantage to cross blood brain barrier and block central presynaptic adrenergic receptors

JAMA. 2006;295:165-171

Class				
I	Drug	Dose (Parenteral)	Dose (Oral)	Desires plasma levels
	Procainamide	Bolus: 10 mg/kg IV over 20 min Infusion: up to 2-3 g/24 h	Oral : 3-6 g in 3 divided doses	4-12 ug/mL
	Lidocaine	Bolus: 1.0 to 1.5 mg/kg IV, repeat dose of 0.5-0.75 mg/kg IV up to a total dose of 3 mg/kg Infusion: 20 µcg/kg per minute IV	Not recommended	2-6 µg/mL
	Mexilitene	Not recommended	Oral : 200 mg TDS , up to 400 mg TDS	0.6-1.7 µg/mL
II				
	Propranolol	Bolus: 0.15 mg/kg IV over 10 min	Oral : 10-40 mg three-four times a day	NA
	Metoprolol	Bolus: 2-5 mg IV every 5 min up to 3 doses in 15 min	Oral : 25 mg by twice a day up to 200 mg a day	NA
	Esmolol	Bolus: 300 to 500 mg/kg IV for 1 min Infusion: 25-50 mg/kg per minute up to a maximum dose of 250 mg/kg per minute (titration every 5-10 min	Not recommended	NA
III	Amiodarone	Bolus: 150 mg IV over 10 min, up to total 2.2 g in 24 h Infusion: 1 mg/min for 6 h, then 0.5 mg/min for 18 h	Loading dose (oral) : 800 mg BD until 10 g total Maintenance dose: 200-400 mg daily	1.0-2.5 µg/mL
	Sotalol	Not recommended	Oral : 80 mg BD, up to 160 mg BD	1-3 µg/mL (not of great value, usually monitored by QT prolongation with indication to reduction/discontinuation if prolongation > 15%-20%)

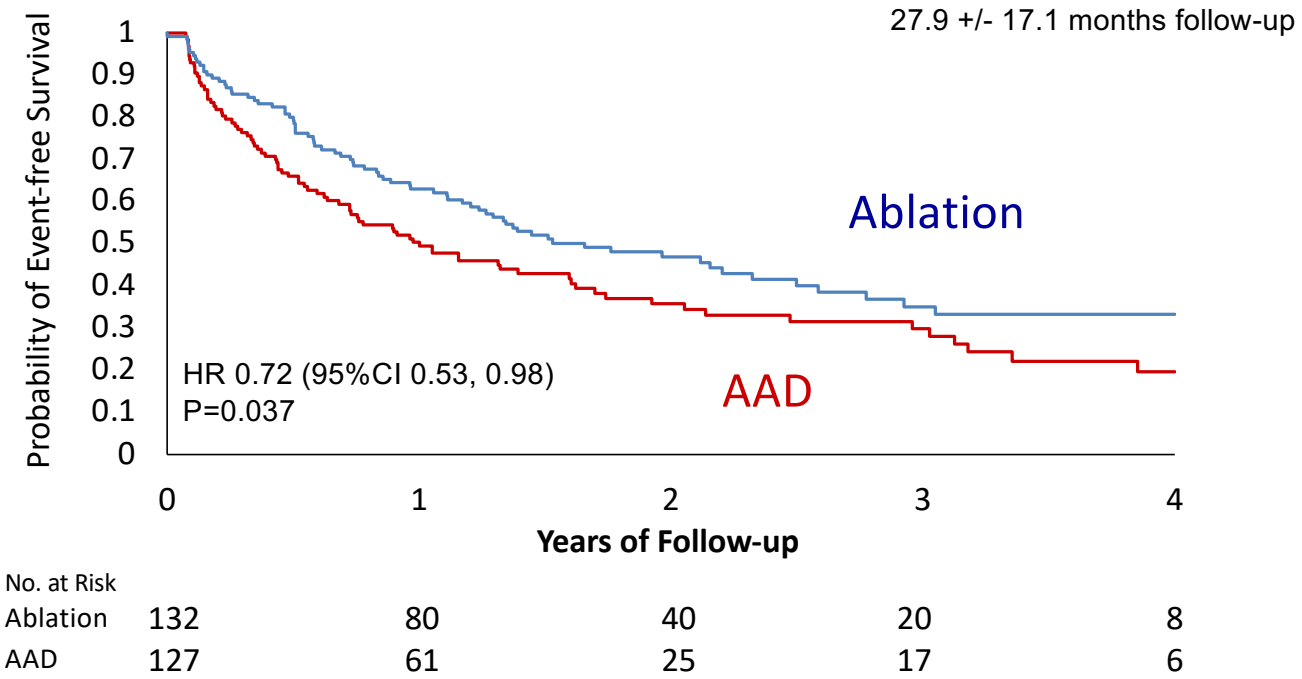
VT Storm Management

1. Evaluate the rhythm and Stabilize hemodynamically
2. Evaluate for triggers. This patient has monomorphic VT, therefore scar-related.
3. Antiarrhythmic therapy: **lidocaine best**

4. Overdrive pace in the atrium (increase lower pacing rate to 80bpm)
5. Sedate the patient: remove the sympathetic drive
6. Neuraxial modulation: stellate ganglion blockage (bupivacaine)
7. Ablation …

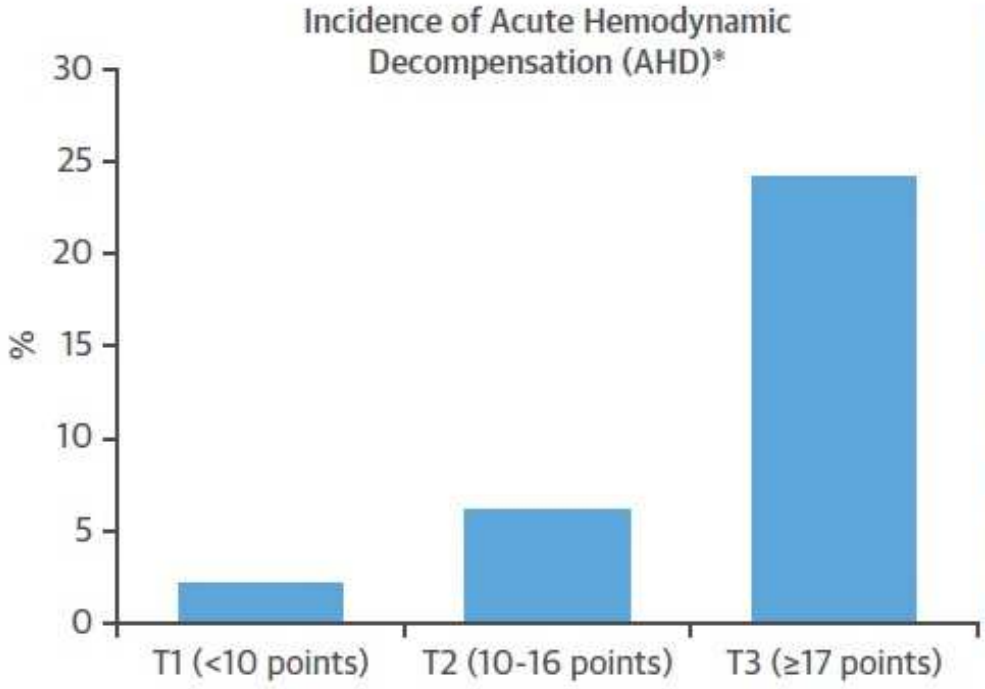
VT ablation in ischemic cardiomyopathy: earlier the better once failure of AAD. VANISH Study: ischemic cardiomyopathy ablation study

Results: Primary Outcome: Death, VT Storm, Appropriate Shock



Predictors of AHD – *PAAINESD* Score

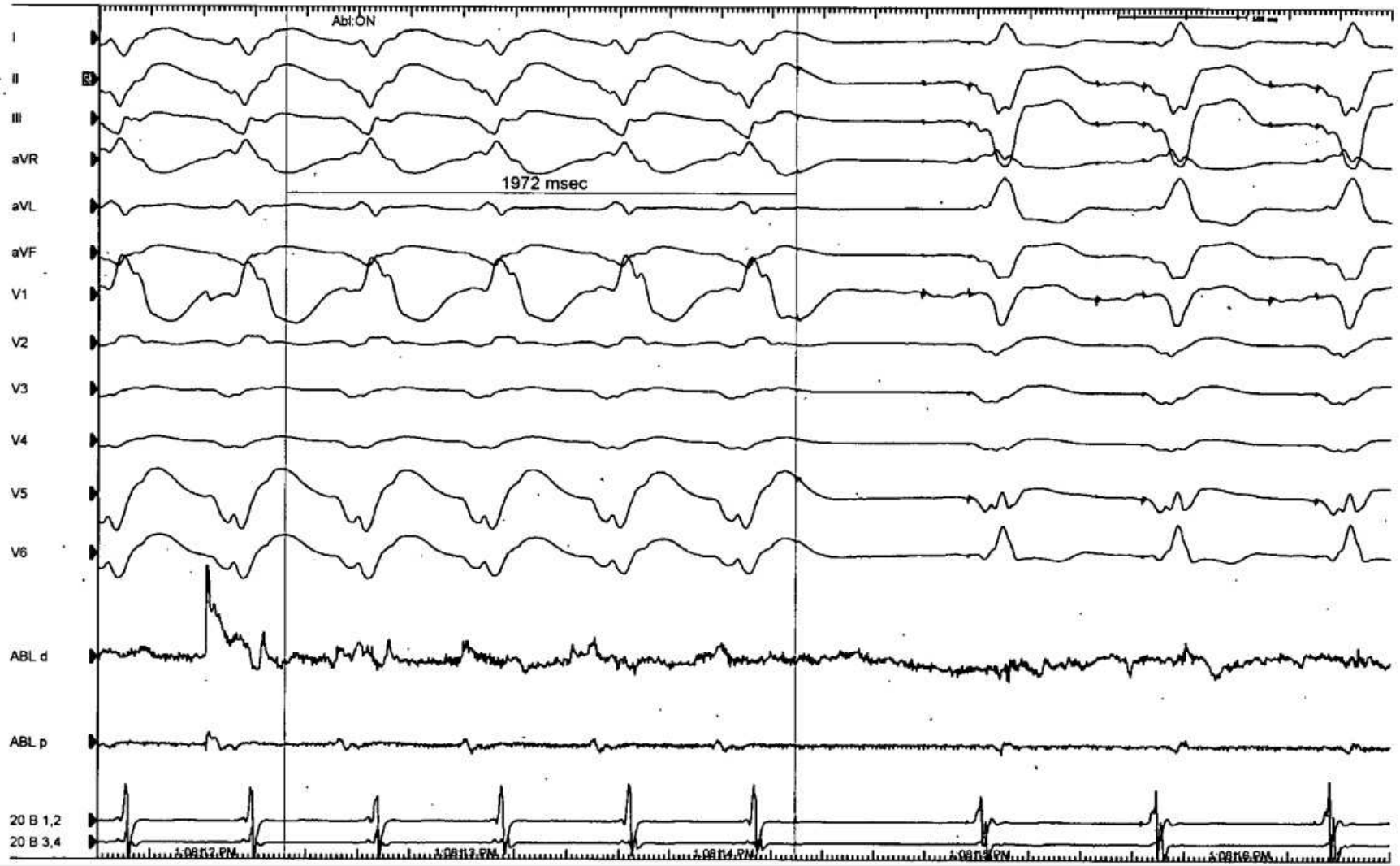
N = 193 patients w/ scar-related VT

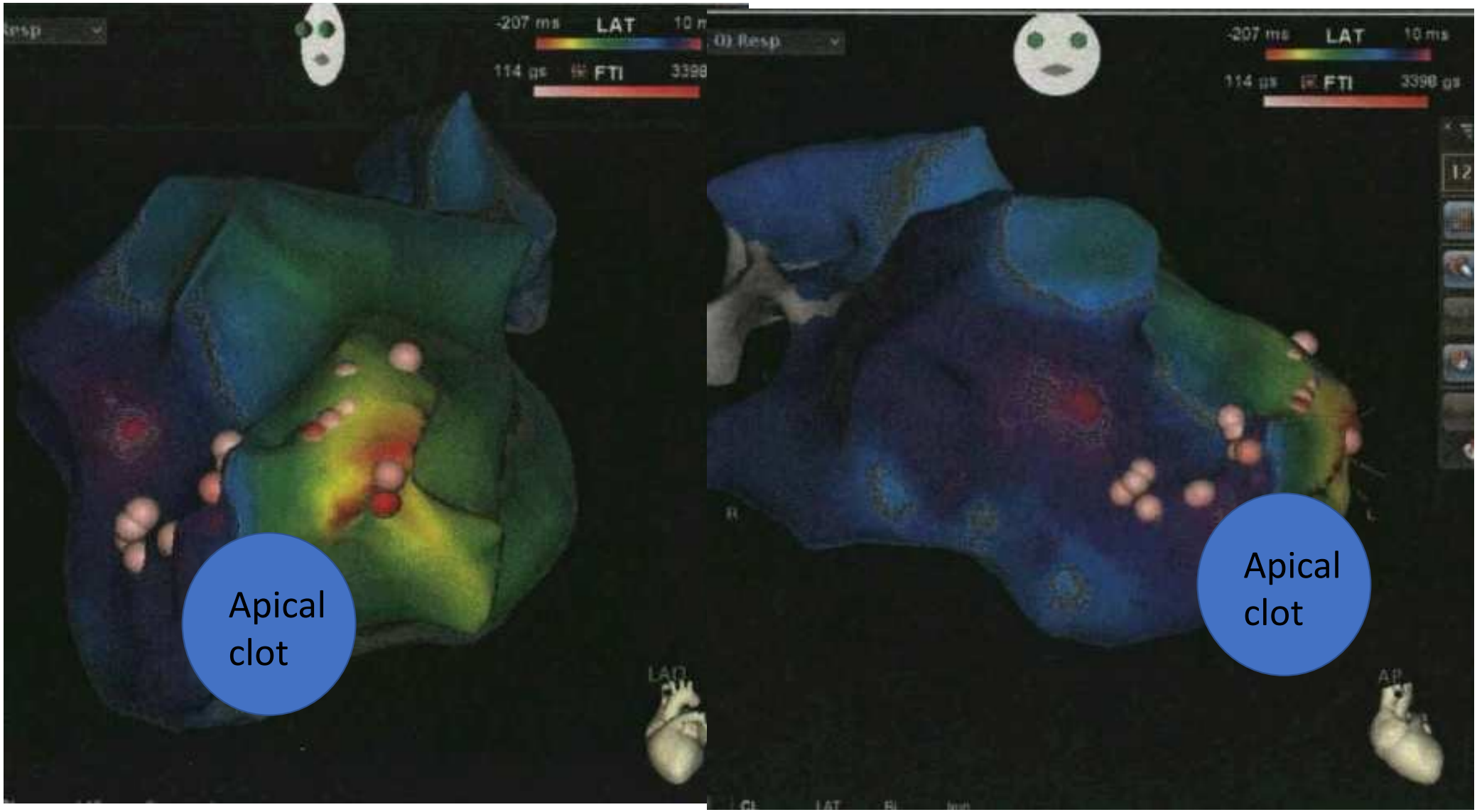


*Sustained hypotension (SBP <80-90 mm Hg) despite increasing doses of vasopressors and requiring mechanical hemodynamic support or procedure discontinuation.

PAAINESD RISK SCORE	
VARIABLE	SCORE
Pulmonary disease [chronic obstructive] – COPD	5
Age >60 years	3
Anesthesia [general]	4
Ischemic cardiomyopathy	6
NYHA class III or IV	6
Ejection fraction <25%	3
Storm [VT]	5
Diabetes mellitus	3

Termination of VT during ablation

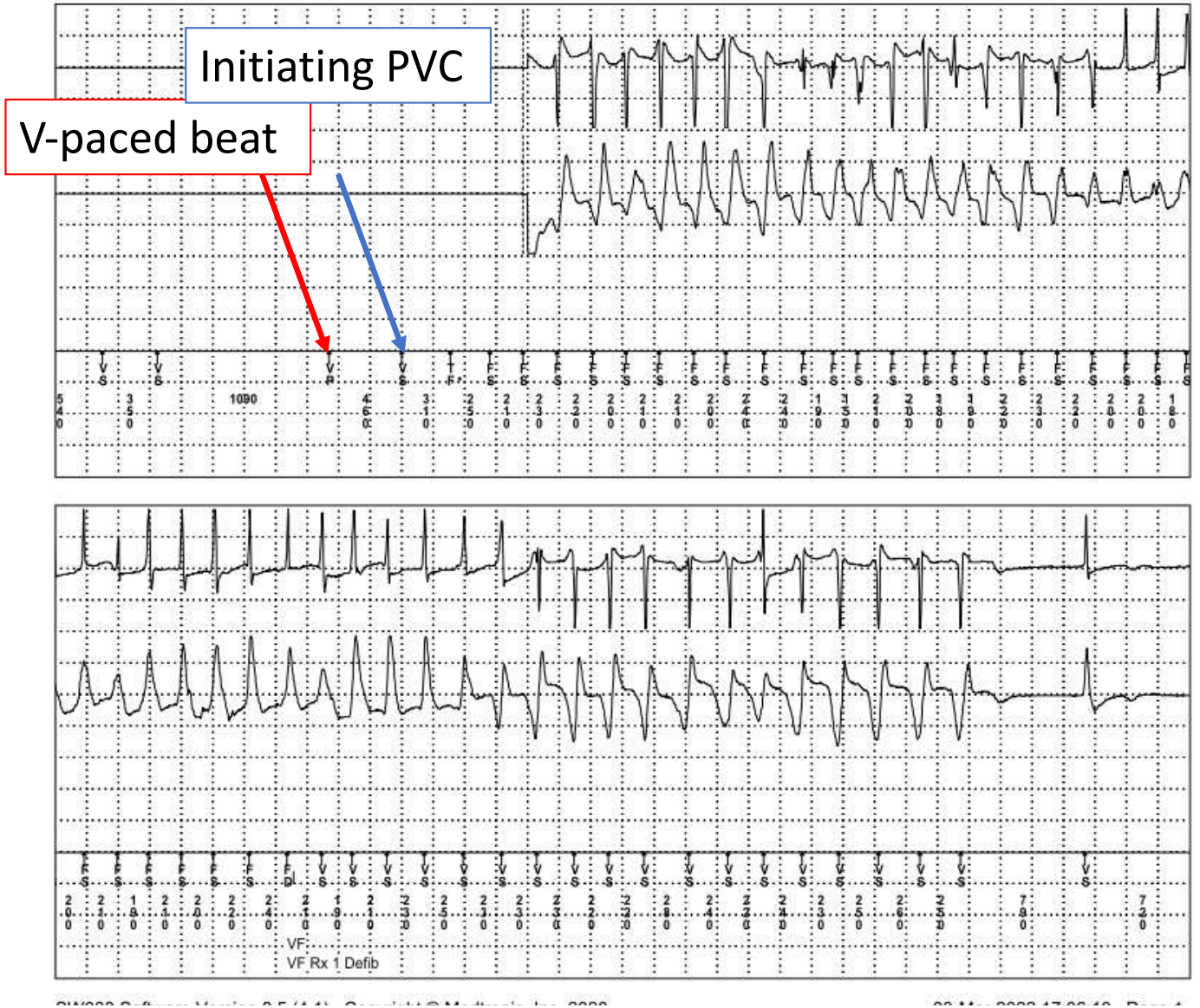


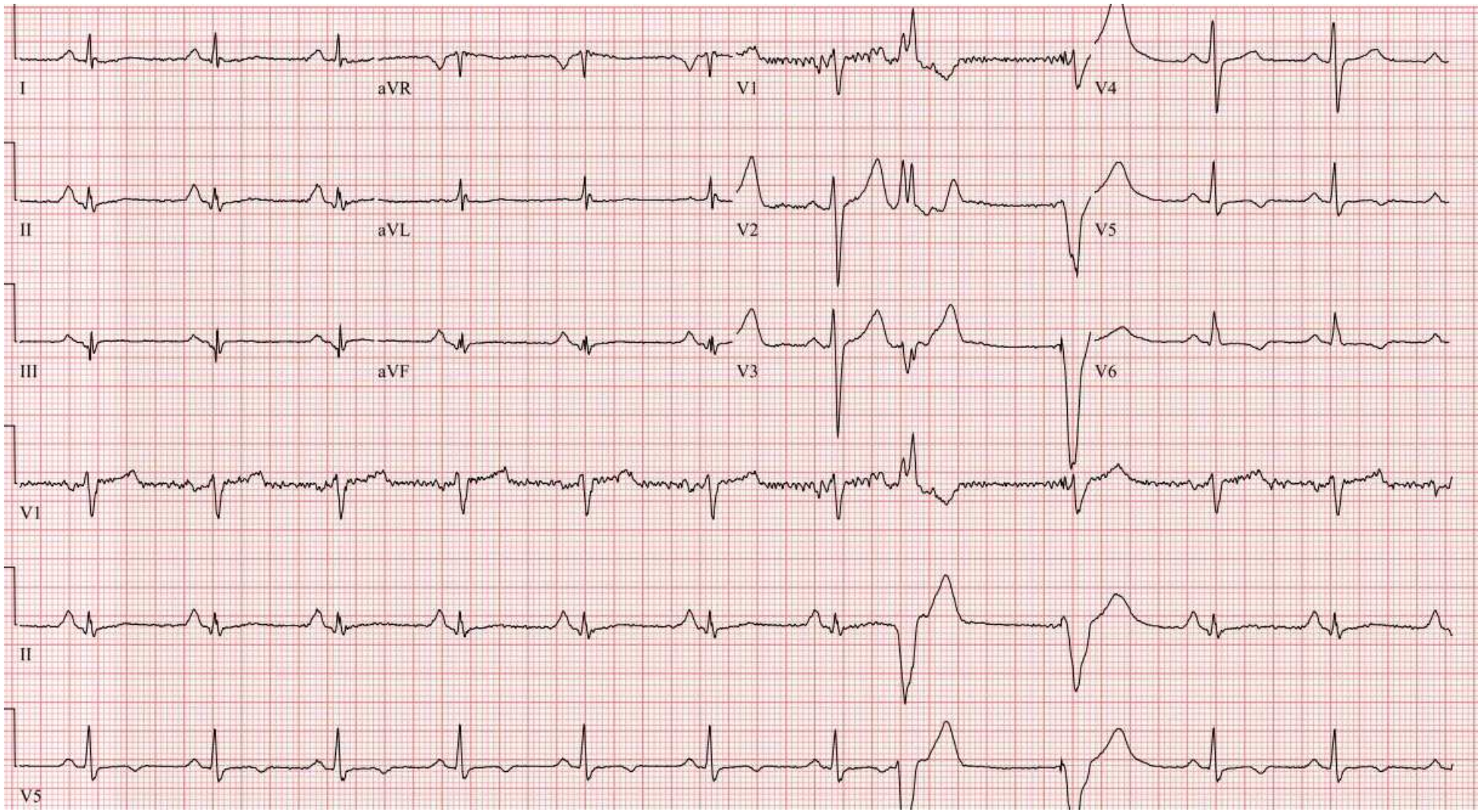

-207 ms
LAT
114 ms
FTI
Apical clot
Apical clot

VT ablation: **nonischemic** cardiomyopathy: this will make a difference

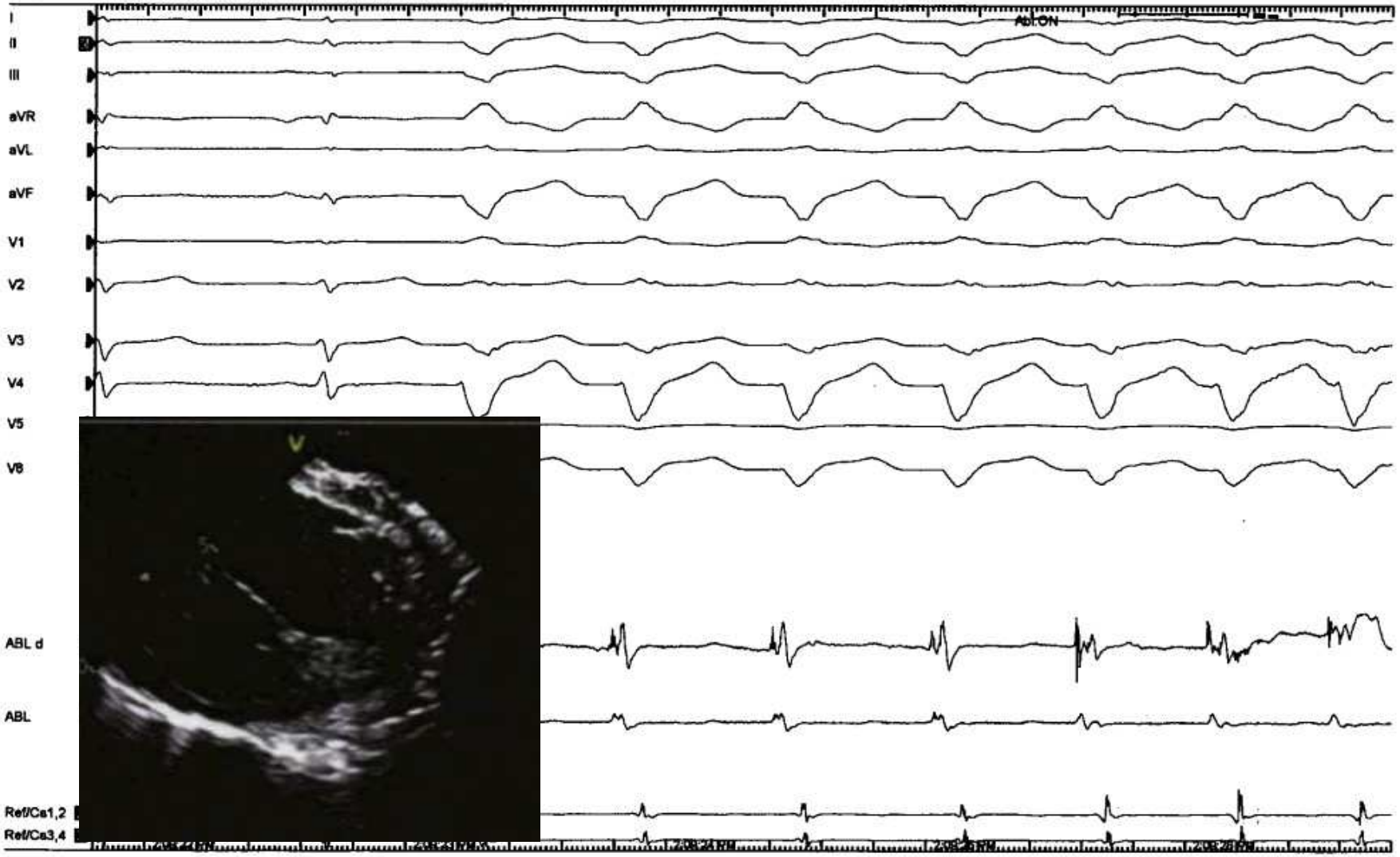
- Patients presenting with polymorphic VT/VF: catheter ablation is indicated when there is a consistent trigger mechanism for the episodes (e.g. recurrent PVC).
  - In these patients, pharmacological control is challenging.

Single chamber VVI ICD in a nonischemic dilated CM EF 10%




I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

Again, PVC#1 morphology (neg I, inf axis, double transition at V3)



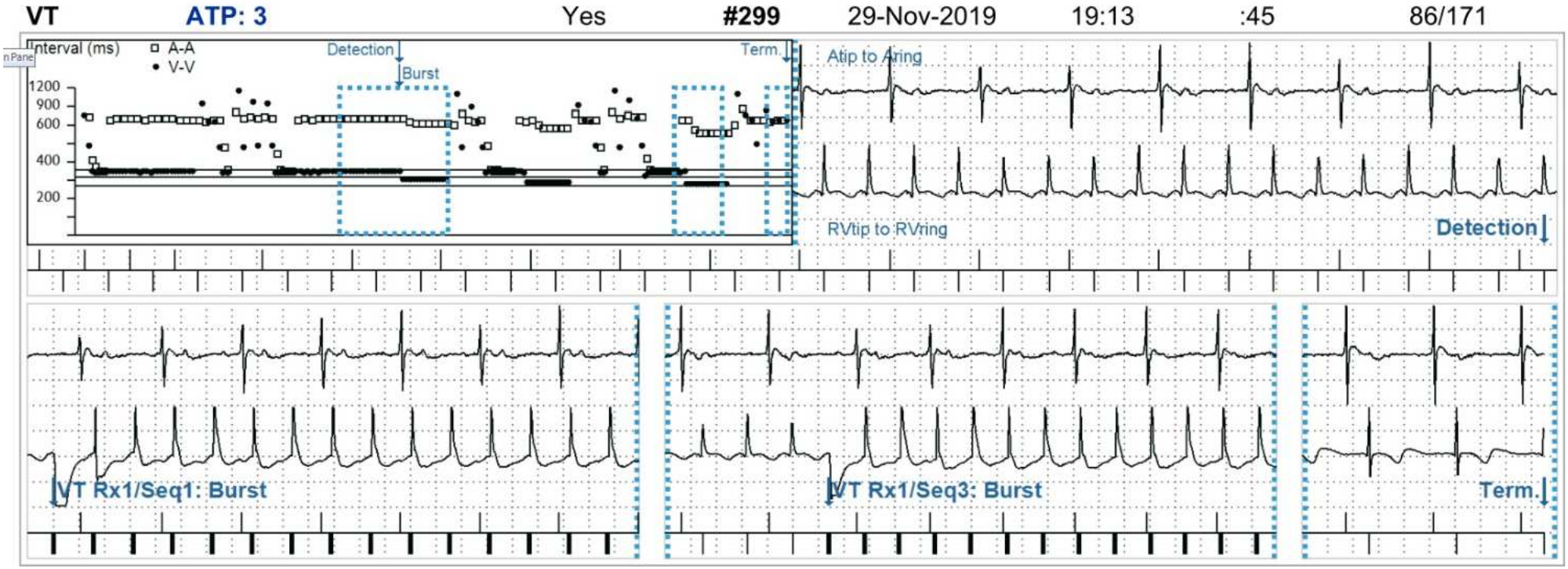
Case 1: 2020: AB

3 years later: 58 years-old

Presents in recurrent sustained VT requiring ICD shocks despite sotalol

Switched to oral amiodarone, but recurrent therapies all from the Lv apex – site of chronic clot…

VT



ATP

ATP

ATP Terminates

Parameter Summary

Mode	AAI<=>DDD	Lower Rate	50 bpm	Paced AV	350 ms
Mode Switch	171 bpm	Upper Track	100 bpm	Sensed AV	350 ms
		Upper Sensor	100 bpm		

Detection		**Rates**	**Therapies**
AT/AF	Monitor	>171 bpm	All Rx Off
VF	On	>188 bpm	ATP During Charging, 35J x 6
FVT	via VF	188-222 bpm	Burst(3), 35J x 5
VT	On	167-188 bpm	Burst(10), Ramp(8), 35J x 4

Enhancements On: VT Monitor, AF/Afl, Sinus Tach, 1:1 SVT, Wavelet, TWave, Noise(Timeout)

Once starting on amiodarone (or sotalol) – must lower the VT detection and therapy rates by at least 20-30bpm because these antiarrhythmics will slow the arrhythmia.

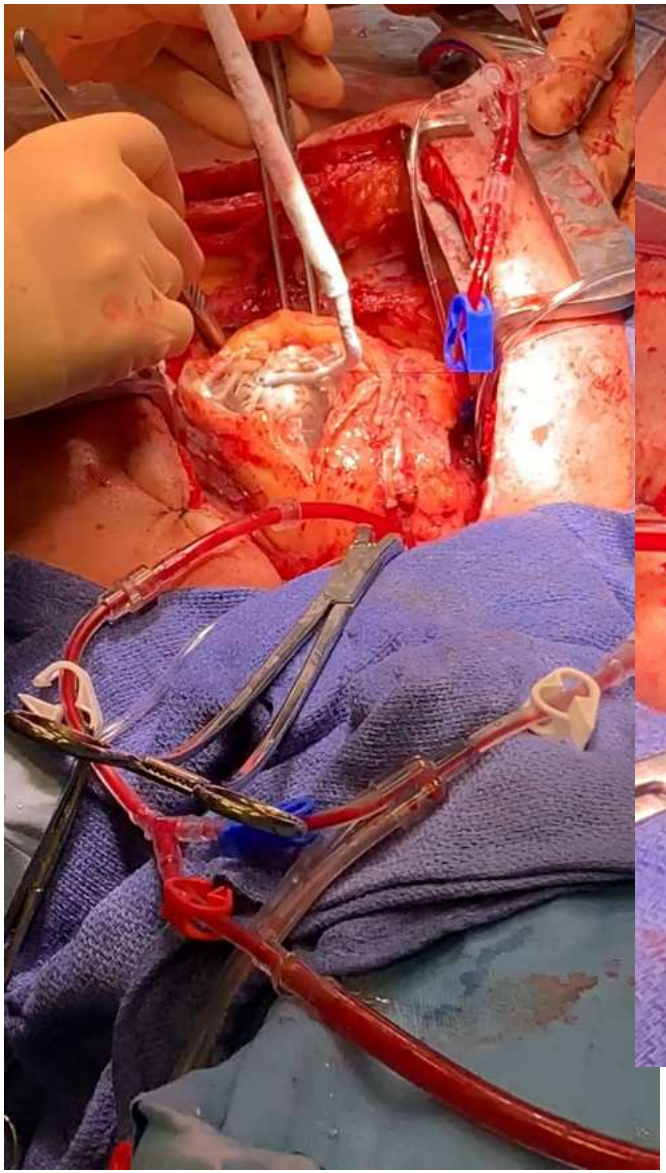
Arrhythmia Episode List: 02-Sep-2019 23:26:05 to 30-Nov-2019 11:52:52

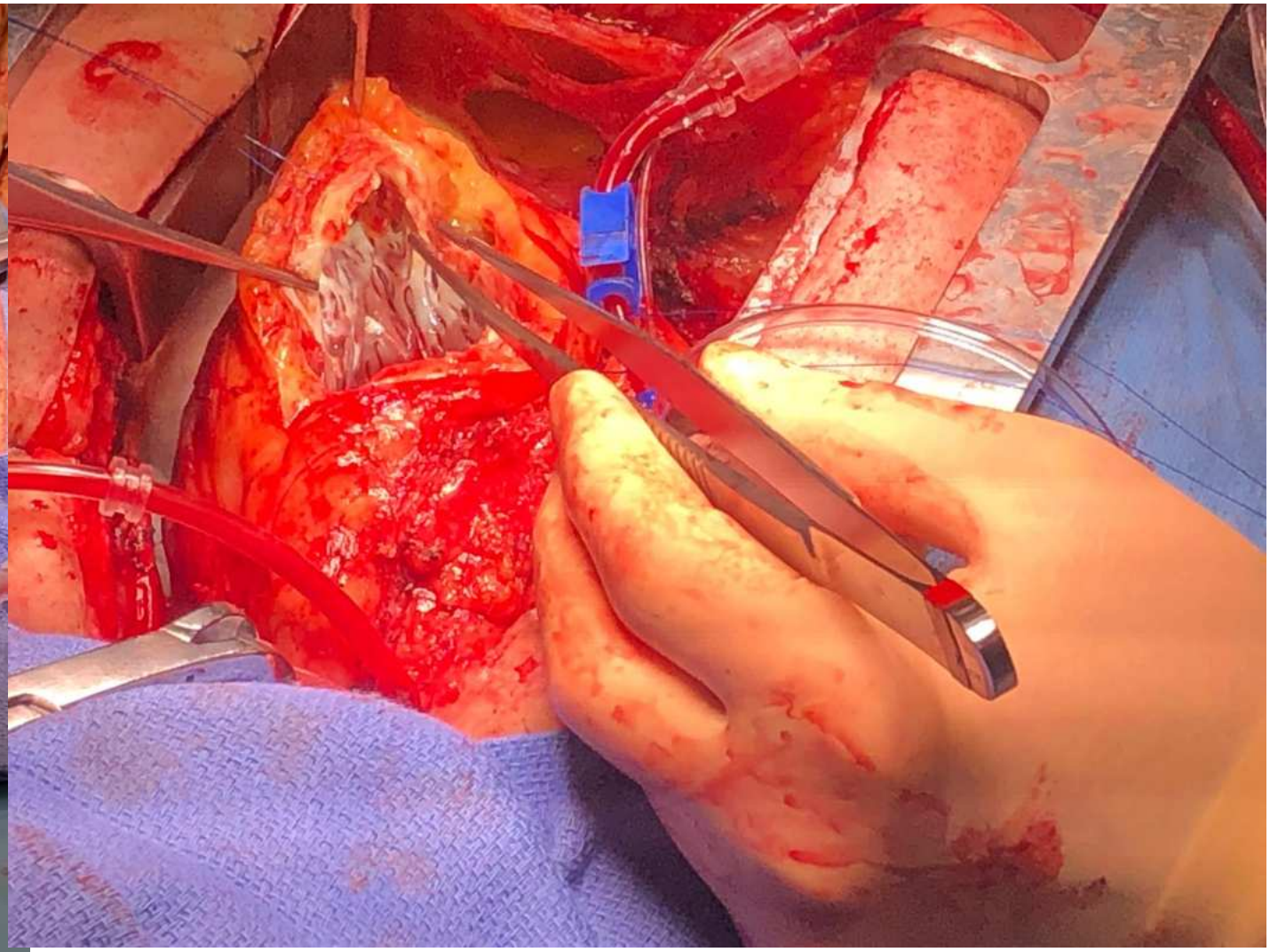
All collected episodes.

Type	ATP Seq	Shocks	Success	ID#	Date	Time hh:mm	Duration hh:mm:ss	Avg bpm A/V	Max bpm A/V	Activity at Onset
VT	2		Yes	306	29-Nov-2019	23:15	:29	171/176	176/176	Rest
VT-NS				305	29-Nov-2019	23:15	:11	130/176		Rest
VT-NS				304	29-Nov-2019	23:15	:11	122/174		Rest
VT	4		Yes	303	29-Nov-2019	22:52	:50	90/176	171/176	Rest
VT-NS				302	29-Nov-2019	22:52	:12	101/174		Rest
VT-NS				301	29-Nov-2019	22:52	:07	91/174		Rest
VT-NS				300	29-Nov-2019	19:35	:08	88/171		Rest
VT	3		Yes	299	29-Nov-2019	19:13	:45	86/171	171/171	Active
VT-NS				298	29-Nov-2019	19:13	:10	90/172		Active
VT-NS				297	29-Nov-2019	19:12	:10	89/171		Active
VT-NS				296	29-Nov-2019	19:11	:10	87/171		Active

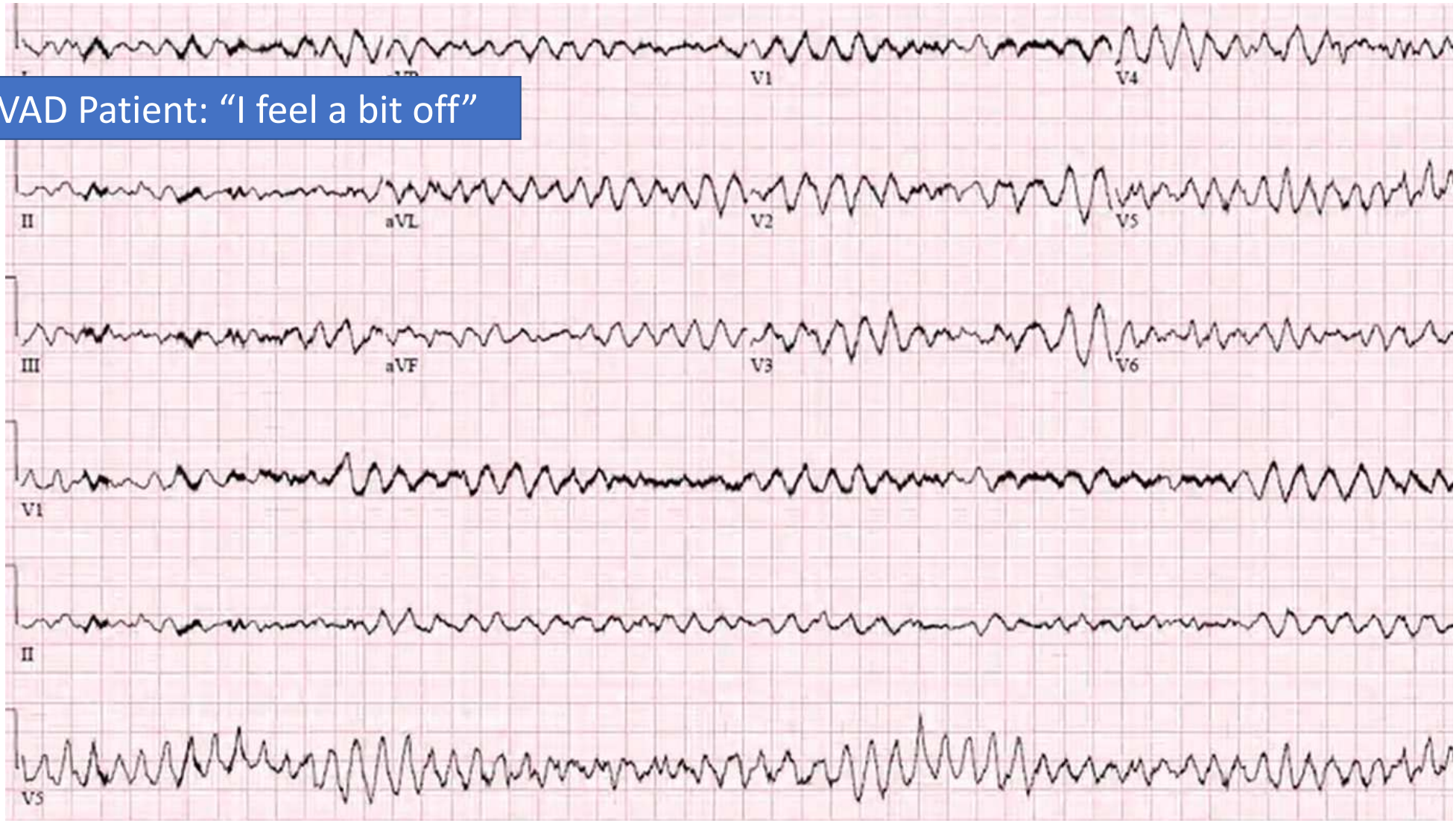
Case 1: 2020: AB

Persistent clot within the LV apex





LVAD Patient: “I feel a bit off”



Question

Do Ventricular arrhythmias worsen prognosis after LVAD implant?

A. No – no major influence on hemodynamics
B. Yes – only the early ventricular arrhythmias post LVAD
C. Yes – all of the ventricular arrhythmias post LVAD: early and late

ORIGINAL ARTICLE

A Fully Magnetically Levitated Left Ventricular Assist Device — Final Report

M.R. Mehra, N. Uriel, Y. Naka, J.C. Cleveland, Jr., M. Yuzefpolskaya, C.T. Salerno, M.N. Walsh, C.A. Milano, C.B. Patel, S.W. Hutchins, J. Ransom, G.A. Ewald, A. Itoh, N.Y. Raval, S.C. Silvestry, R. Cogswell, R. John, A. Bhimaraj, B.A. Bruckner, B.D. Lowes, J.Y. Um, V. Jeevanandam, G. Sayer, A.A. Mangi, E.J. Molina, F. Sheikh, K. Aaronson, F.D. Pagani, W.G. Cotts, A.J. Tatooles, A. Babu, D. Chomsky, J.N. Katz, P.B. Tessmann, D. Dean, A. Krishnamoorthy, J. Chuang, I. Topuria, P. Sood, and D.J. Goldstein, for the MOMENTUM 3 Investigators*

ABSTRACT

Ventricular Arrhythmias as Adjudicated Cause of Death over 2 years follow-up:

- Centrifugal flow pump – 2 of 98 deaths
- Axial flow pump – 1 of 103 deaths

Cancer: 2 centrifugal – 0 axial

Driveline/power/battery issues: 6 centrifugal – 2 axial

Mehra et al. MOMENTUM 3 Investigators NEJM 2019;380:1618-

Table S5. Adjudicated Causes of Death (Per Protocol Population)

Adjudicated Cause of Death	Centrifugal-flow pump (n=515)	Axial-flow pump (n=505)	Total
Cardiopulmonary related			
Cardiac arrest	0	1	1
Heart failure	4	1	5
Pericardial tamponade	0	2	2
Respiratory failure	3	2	5
Right heart failure	31	26	57
Ventricular arrhythmia	2	1	3
Brain related			
Anoxic brain injury	3	0	3
Head trauma	2	0	2
Intracranial Bleed Due to Trauma	2	2	4
Stroke	13	29	42
Bleeding related			
Aortic dissection	0	1	1
Abdominal or gastrointestinal bleeding	1	3	4
Infection related			
Infection or sepsis	14	14	28
Pneumonia	4	2	6
Device related			
Driveline or power cable disconnect*	5	1	6
MPU disconnected from power	1	0	1
Pump stop	0	1	1
Pump thrombosis	1	8	9

Ventricular Arrhythmias (VAs) post LVAD are associated with increased morbidity and risk of death

- Pain/trauma of ICD shocks
- RV failure from VA (45% vs 23% incidence early post-op) and from multiple ICD shocks

Decreased CO and frequent need for RVAD, inhaled pulmonary vasodilators, inotropes

MOMENTUM 3: right heart failure most common cause of death

Garan AR et al. J Heart Lung Tranplsnat 2015; 34(12):1611-

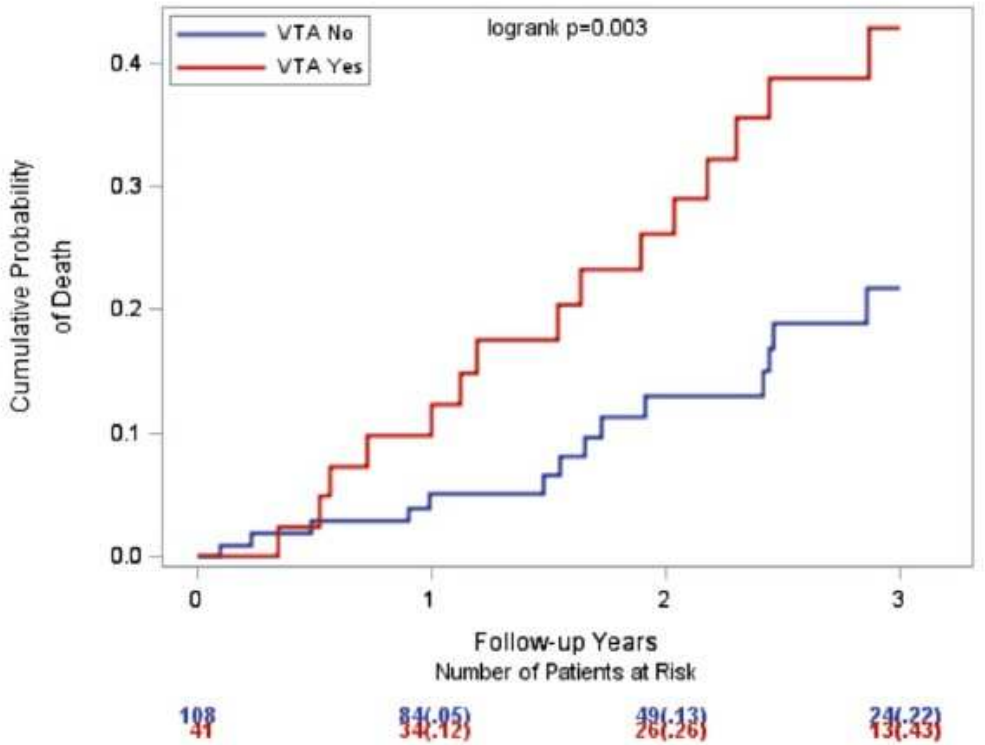
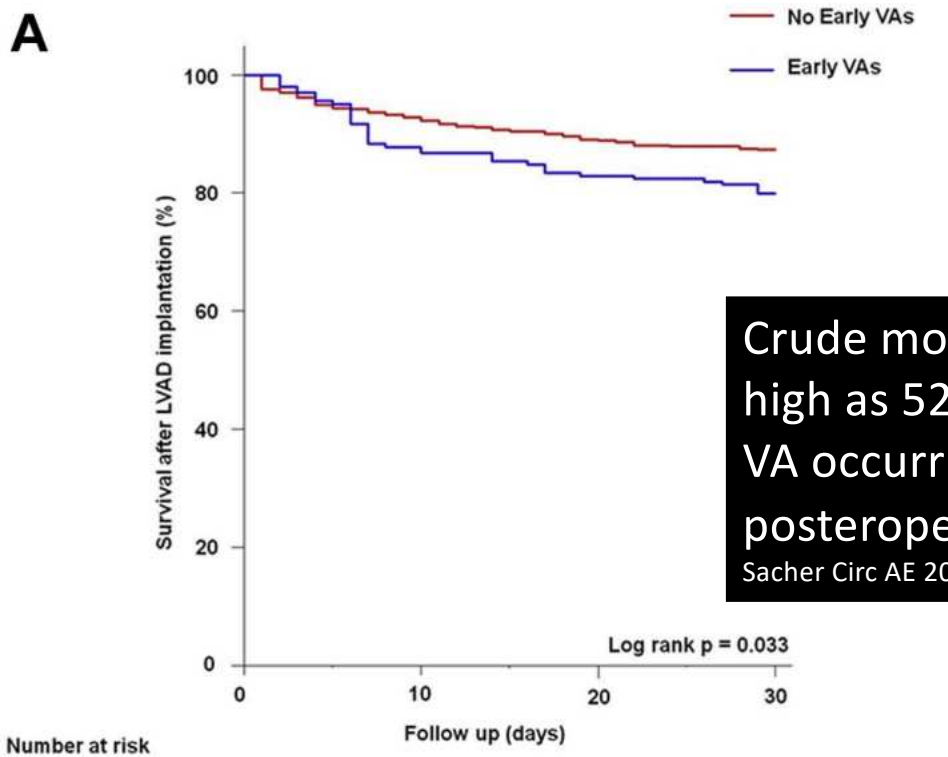


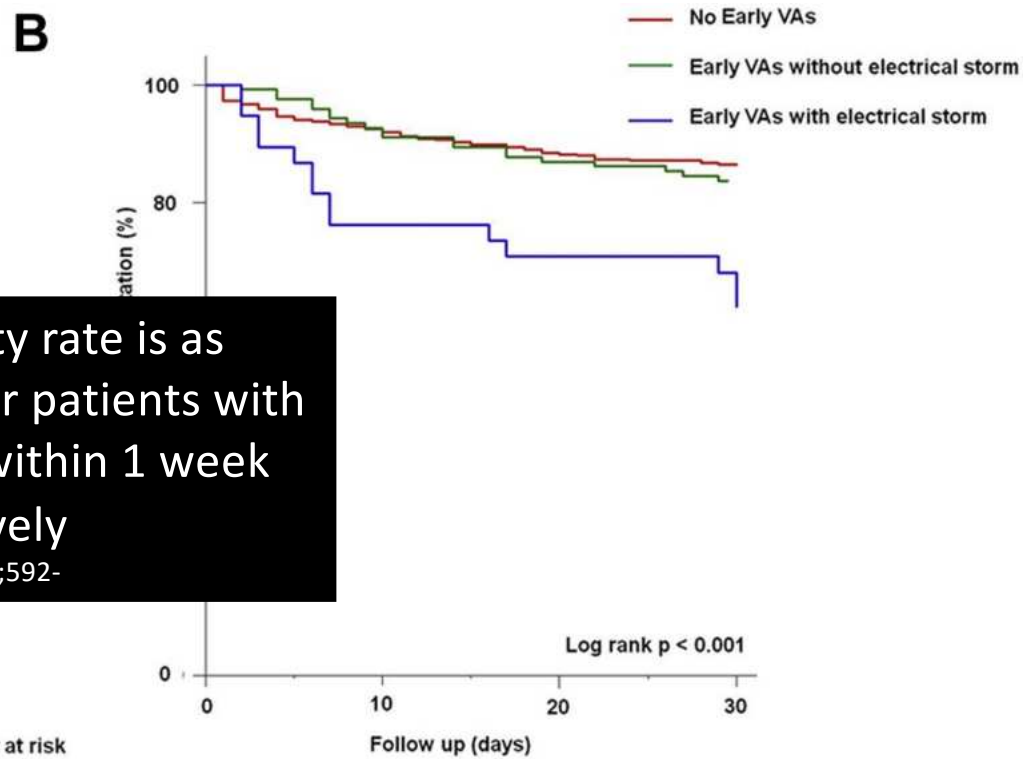
Figure 2 Kaplan-Meier estimates of the cumulative probability of death after LVAD implantation, dichotomized by a history of VTA. LVAD = left ventricular assist device; VTA = ventricular tachyarrhythmia.

Yoruk et al. Heart Rhythm 2016; 13(3):1052-

VAs <u>early</u> post LVAD portend a negative prognosis acutely



Number at risk				
Early VAs	162	144	134	129
No early VA	490	450	429	419



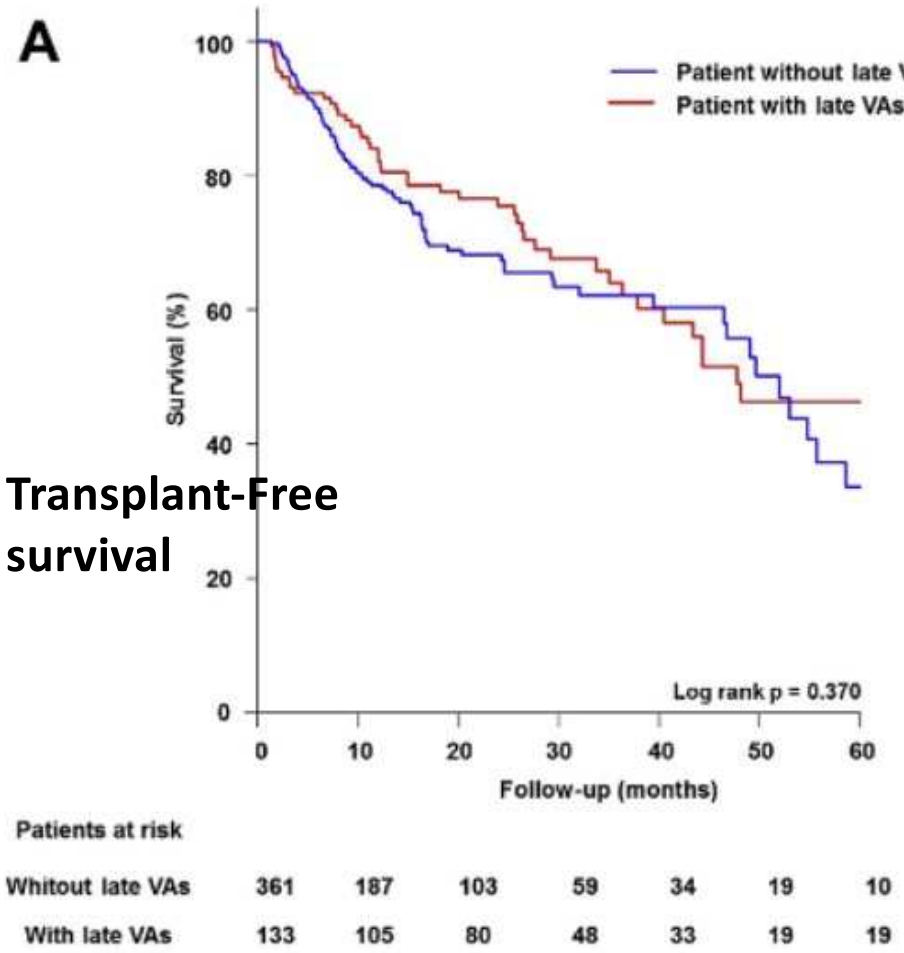
Number at risk				
Early VAs and AS	39	31	25	24
Early VAs and no AS	123	116	108	104
No early VA	490	450	429	417

Crude mortality rate is as high as 52% for patients with VA occurring within 1 week posteroperatively

Sacher Circ AE 2015 8(3);592-

Galand V et al. JACC EP 2019;5:944-

Overall risk of death with late VAs is less clear



Transplant-Free survival

	Late VAs	No Late VAs
Transplant (187/494)	32.2%	39.9%
Death (151/494)	35.3%	28.8%
Cardiovascular death	**61.7%**	**33.7%**
LVAD thrombosis	31%	46%
RV failure	34%	37%
Electrical storm	24%	0%
Non-CV death/unknown	38.3%	66.3%

VAs early post LVAD are common

25% of patients experience early VAs (other observational trials: 13-35%)
57% of patients experience their first early VA during the initial 7 post-operative days

TABLE 2 Multivariable Analysis for Risk Prediction of Early VAs

	β Coefficient	OR (95% CI)	p Value
Body mass index, kg/m^2	0.028	1.03 (0.99-1.07)	0.186
Heart failure duration, months	0.001	1.00 (0.99-1.00)	0.831
LVEDD prior to LVAD, mm	0.012	1.01 (0.99-1.03)	0.103
VAs prior to LVAD	0.859	2.36 (1.57-3.56)	<0.001
ICD prior to LVAD	0.115	1.12 (0.69-1.82)	0.639
Total bilirubin prior to LVAD, mmol/l	0.006	1.01 (1.00-1.01)	0.064
Intra-aortic pump balloon prior to LVAD	−0.275	0.76 (0.33-1.77)	0.524
Surgery combined with LVAD	0.561	1.75 (1.05-2.93)	0.033
Temporary right ECLS during LVAD surgery	−0.739	0.48 (0.24-0.95)	0.035
AF post-LVAD in ICU	0.293	1.34 (0.91-1.97)	0.137

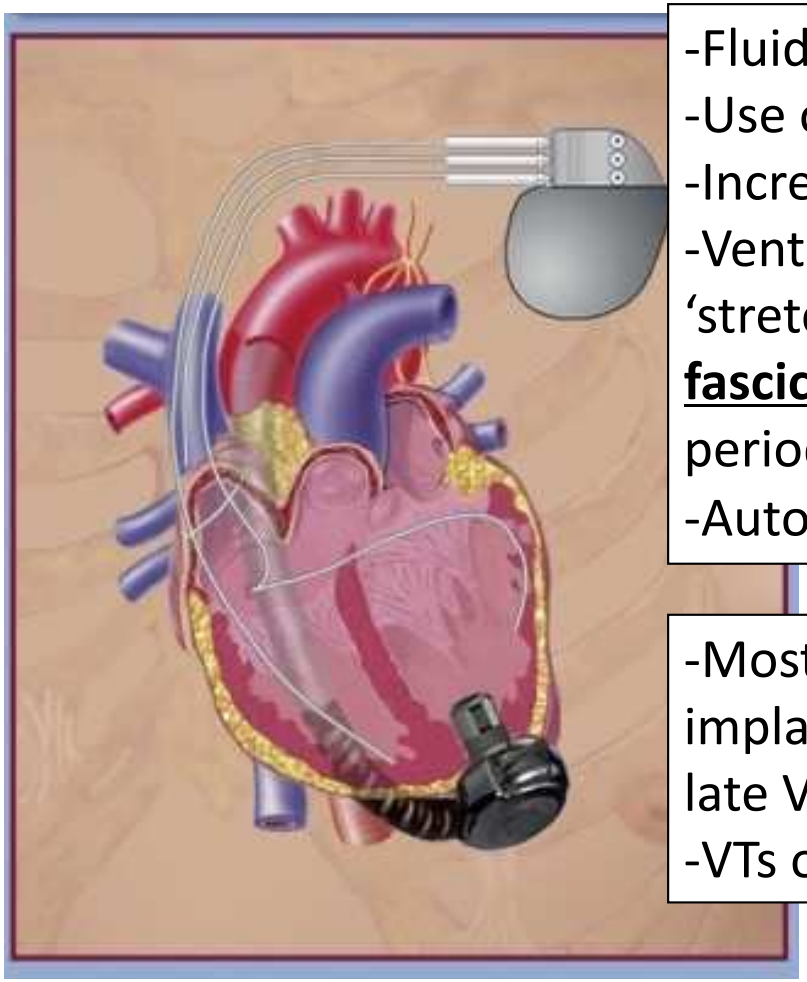
-The apical cannula of the LVAD was incriminated as the substrate of early VAs, but in fact the pre-existing scar is really the main substrate for early VAs.

-Temporary right extracorporeal life support decreased 2-fold risk of VA

Presence of pre-op VA: ie. explained by the underlying arrhythmia substrate

Galand V et al. JACC EP 2019;5:944-

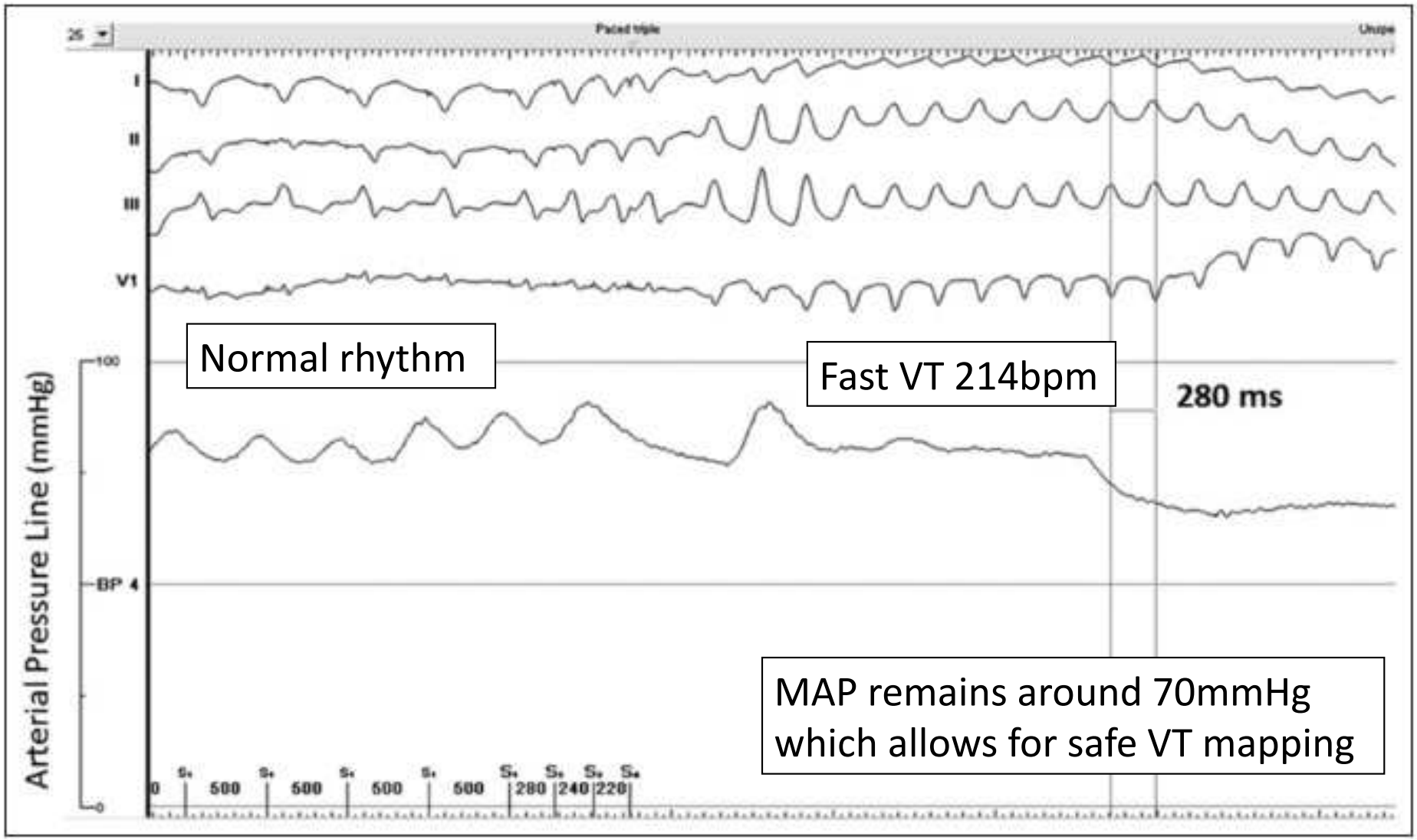
Precipitating Mechanisms and Substrate for Early VAs

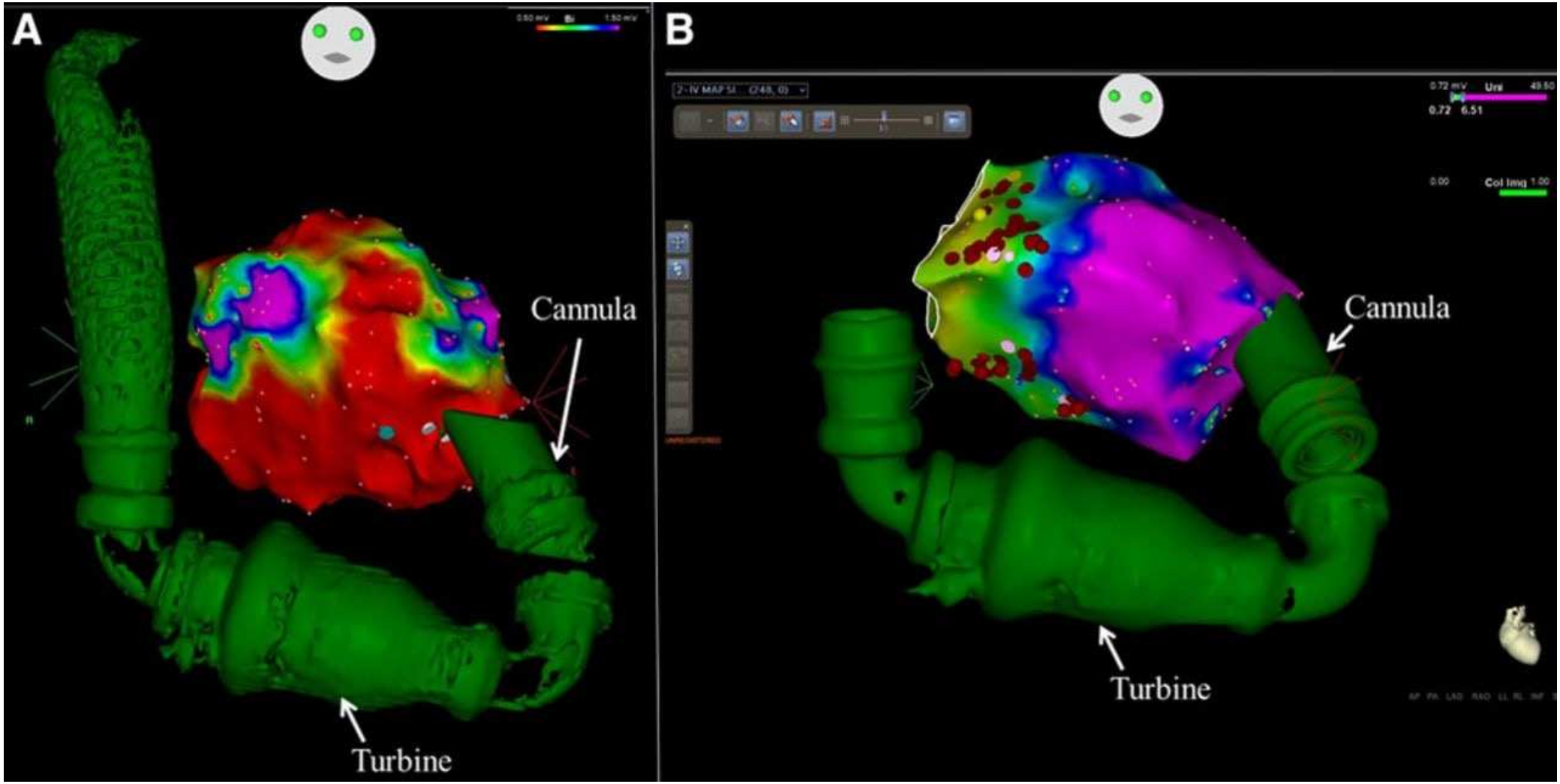


-Fluid and electrolyte shifts
-Use of inotropic drugs
-Increase in QTc interval after LVAD (reported)
-Ventricular unloading: associated with changes in 'stretch' (**tension or distortion of the muscles and fascicles**) that may alter electrical properties (refractory periods or conduction times) particularly in scar areas
-Autonomic nervous system (B-blockers often withheld)

-Most patients with early VAs post LVAD had VA prior to implant (those who did not have pre-existing VAs had late VA occurrence)
-VTs originating near cannula: most occurred after 1 mo.

Sacher Circ AE 2015 8(3);592-


I
II
III
V1
Normal rhythm
Fast VT 214bpm
280 ms
Arterial Pressure Line (mmHg)
100
BP 4
0
500
500
500
500
280
240
220
MAP remains around 70mmHg
which allows for safe VT mapping



Sacher Circ AE 2015 8(3);592-

Epicardial Ablation after LVAD is a Significant Challenge

	Patients, n	ICM, n (%)	CF- LVAD, n (%)	Follow-Up, mo	VTs (average/patient), n	Recurrence, n (%)	Epicardial Ablation, n
Dandamudi et al[154] (2007)	3	2 (66)	0 (0)	4–12	6 (2)	1 (33)	0
Hottigoudar et al[13] (2011)	3	1 (33)	3	2–10	15 (5)	2 (66)	0
Cantillon et al[50] (2012)	21	12 (57)	NR	4.4±3.3	28 (1.3)	7 (33)	0
Herweg et al[155] (2012)	6	4 (66)	4 (66)	7.5±6.9	14 (2.3)	2 (33)	0
Garan et al[156] (2014)	7	5 (71)	7 (100)	5±3.6	19 (2.7)	6 (86)	1
Sacher et al[12] (2015)	34	21 (62)	34 (100)	25±15	110 (3.2)	5 (15)	0
Snipelisky et al[157] (2017)	6	2 (33)	6 (100)	6	18 (3)	5 (83)	1
Moss et al[158] (2017)	21	14 (66)	21 (100)	9	2.5 (2–4.5) per patient	7 (33)	0
Total	101						2

Lessons for VAs post LVAD

-Optimal VA management is unknown

-Occurrence of VA post LVAD implantation (particularly early phase) is deleterious

-Performing ablation before LVAD implantation will likely result in overtreatment
- -high procedural risk in sick population
- -ablation during LVAD implantation: requires precise knowledge of scar location (cryo-ablation)

Galand V et al. JACC EP 2019;5:944-

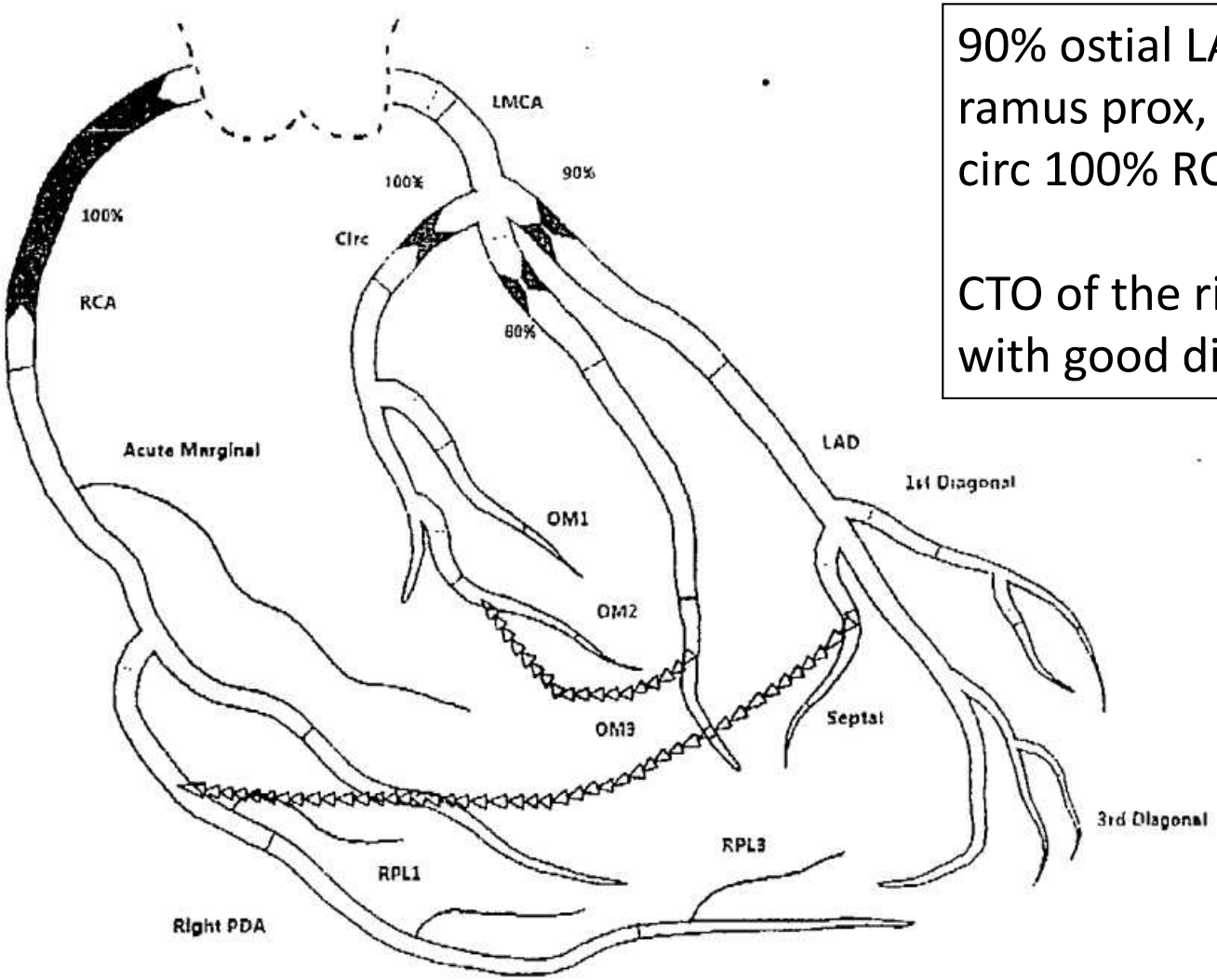
Case 2:

A 62 year-old male presents with dyspnea x 6 months and an ICD shock. NYHA III.

PMHx: CAD EF 25%, DM2, nephrectomy for RCC 2010. Biventricular ICD implanted 2012 and battery change 2017.

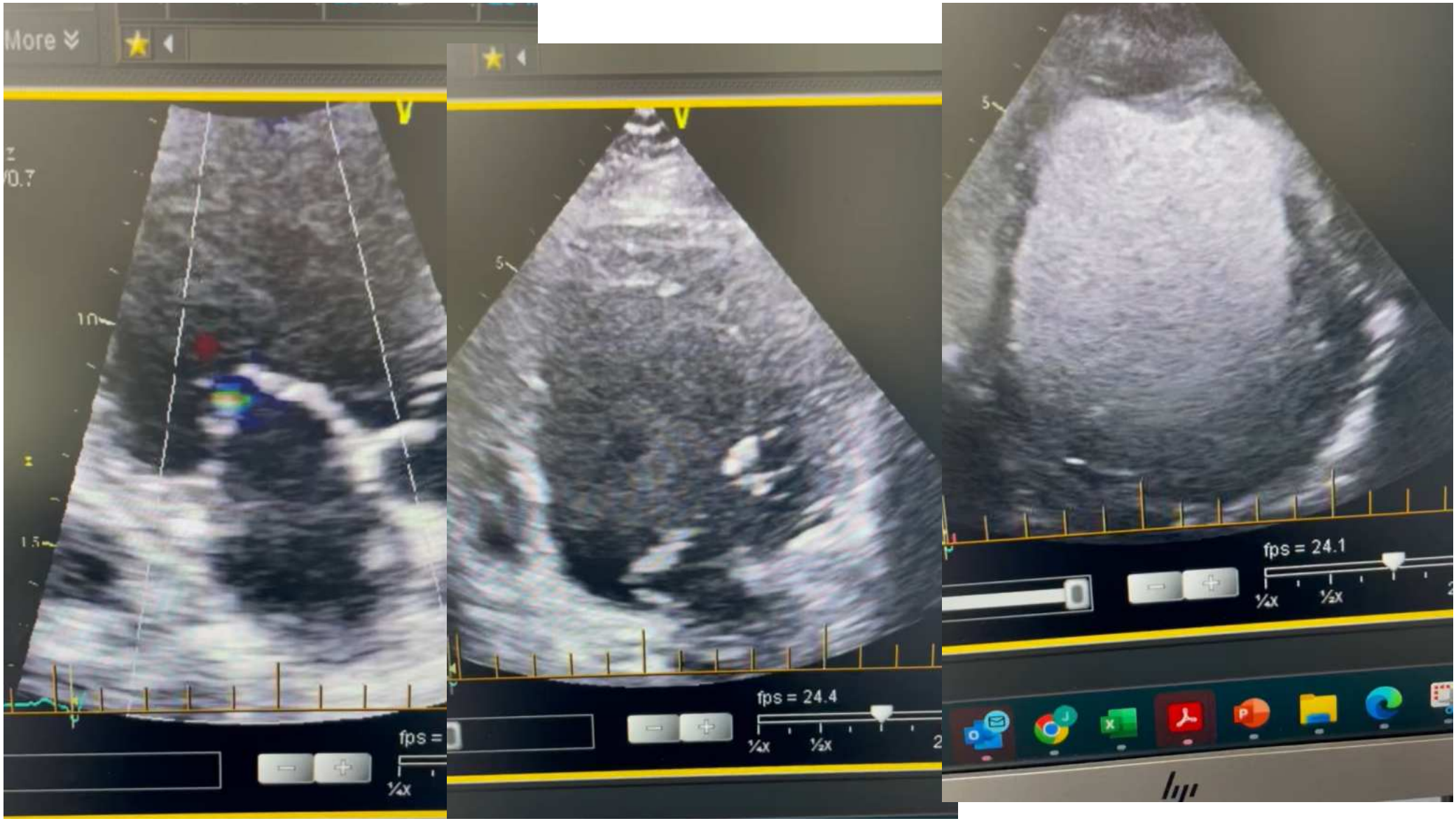
Transthoracic echo: EF 15% with possible thrombus. IV heparin started

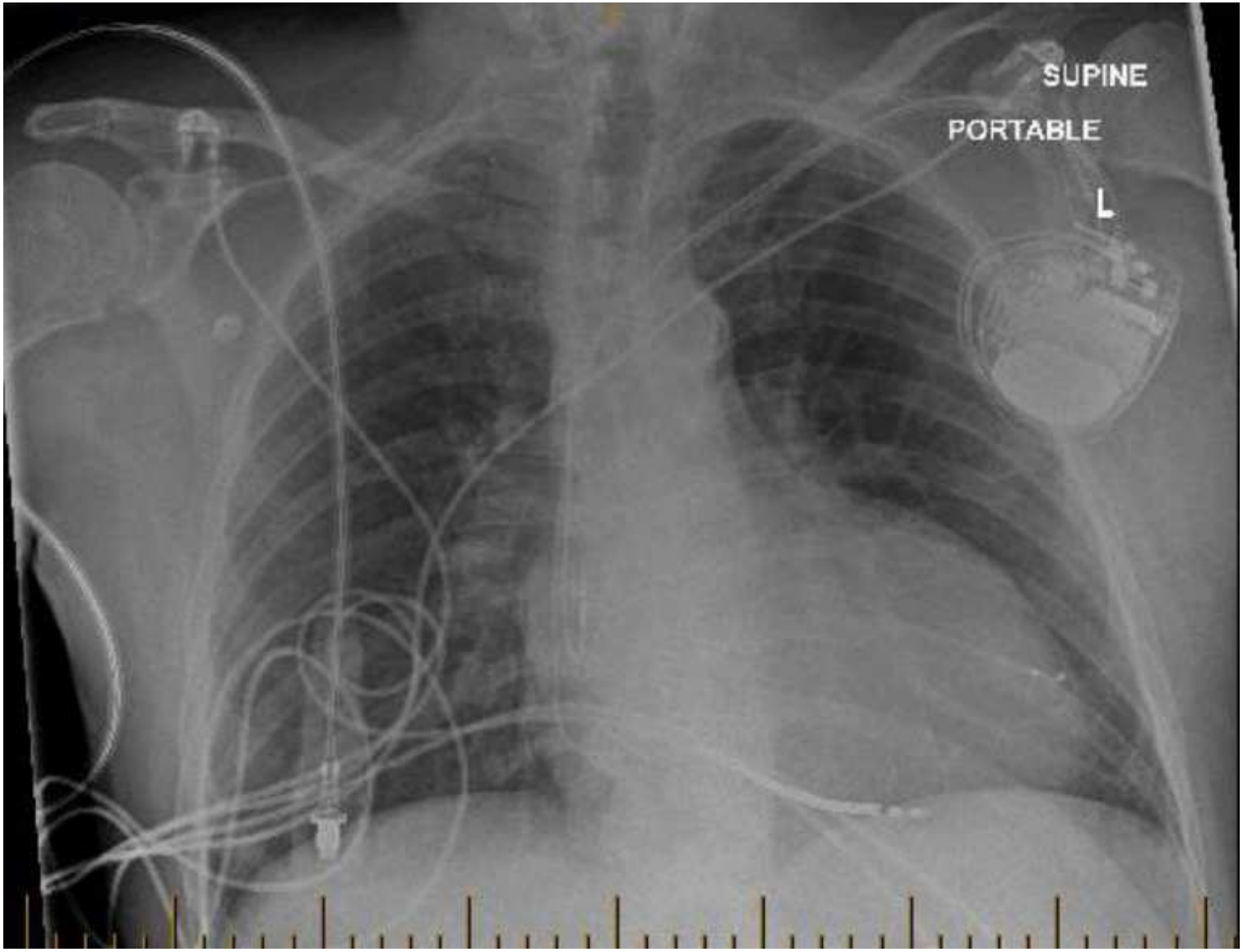
Transferred to our hospital for LVAD evaluation: cardiac arrest on ward, ECMO insertion: 7 days later LVAD implant.



90% ostial LAD 80% ramus prox, 100% prox circ 100% RCA

CTO of the right and circ with good distal beds.


More
fps =
fps = 24.4
fps = 24.1


SUPINE
PORTABLE
L

Case 2: 62M

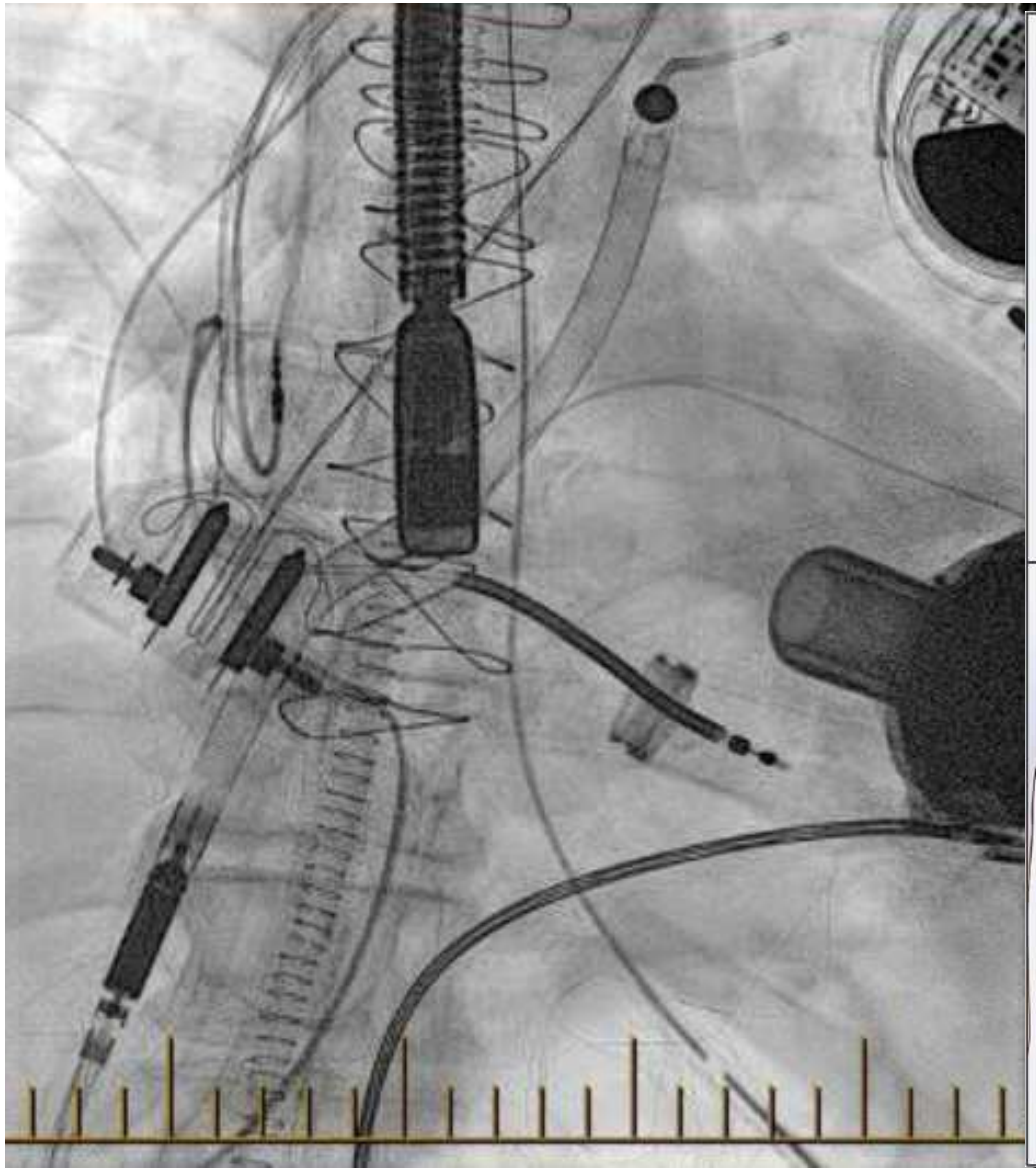
Transthoracic echo: EF 15% with possible thrombus. IV heparin started

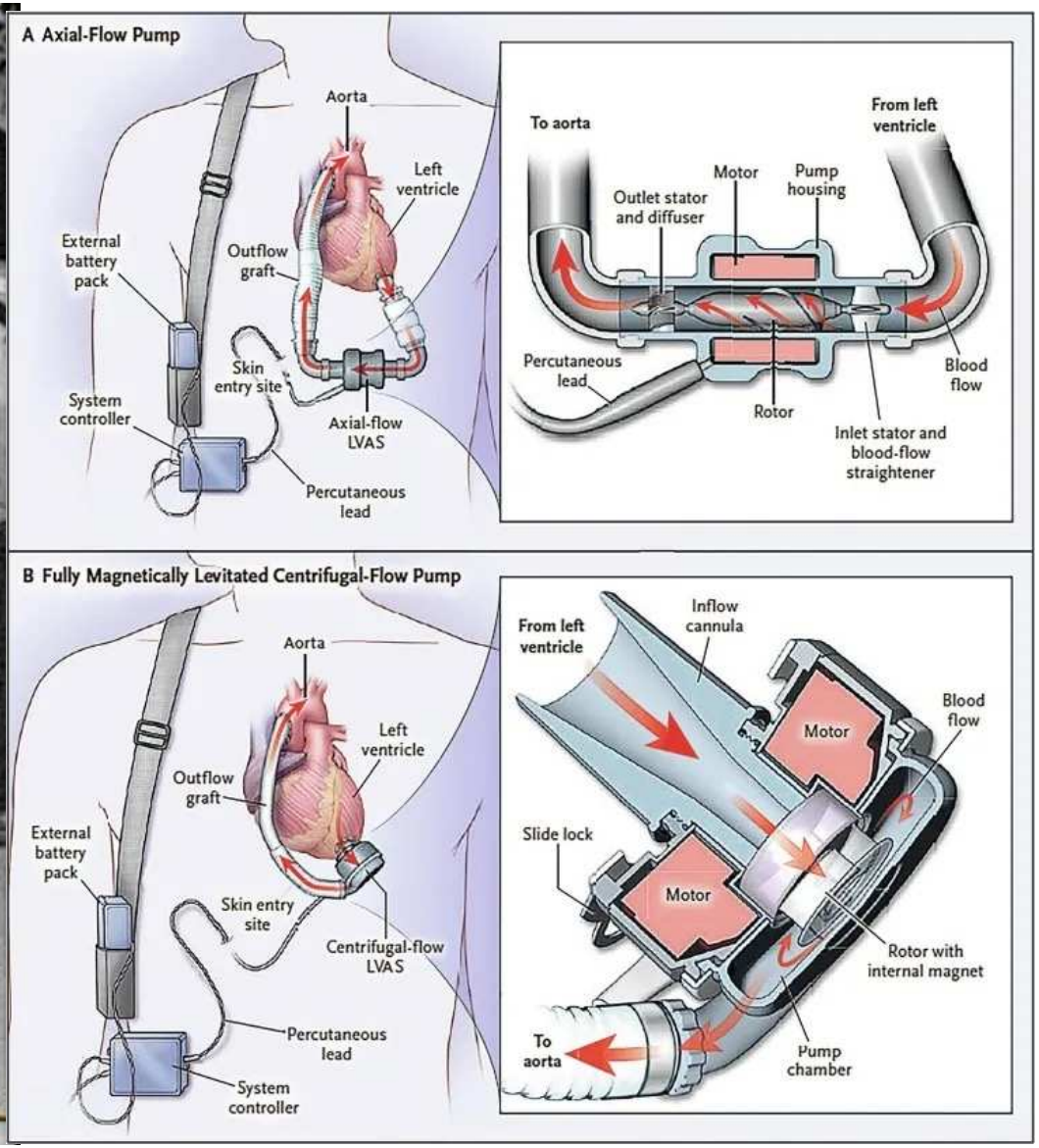
Transferred to our hospital for LVAD evaluation: cardiac arrest on ward, ECMO insertion: 7 days later LVAD implant.

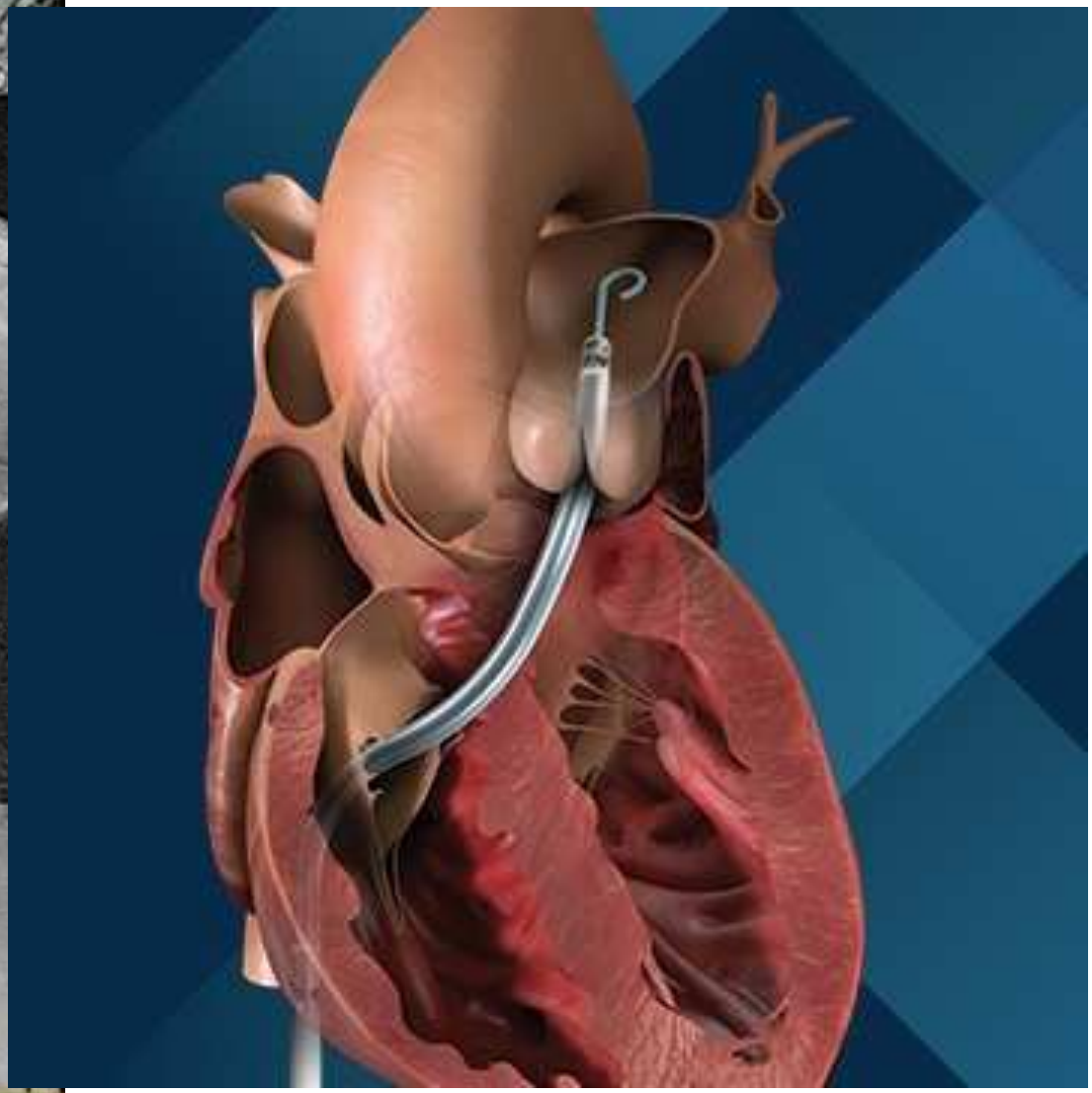
During LVAD surgery, developed RV failure with LVAD flows of 1.5L/min

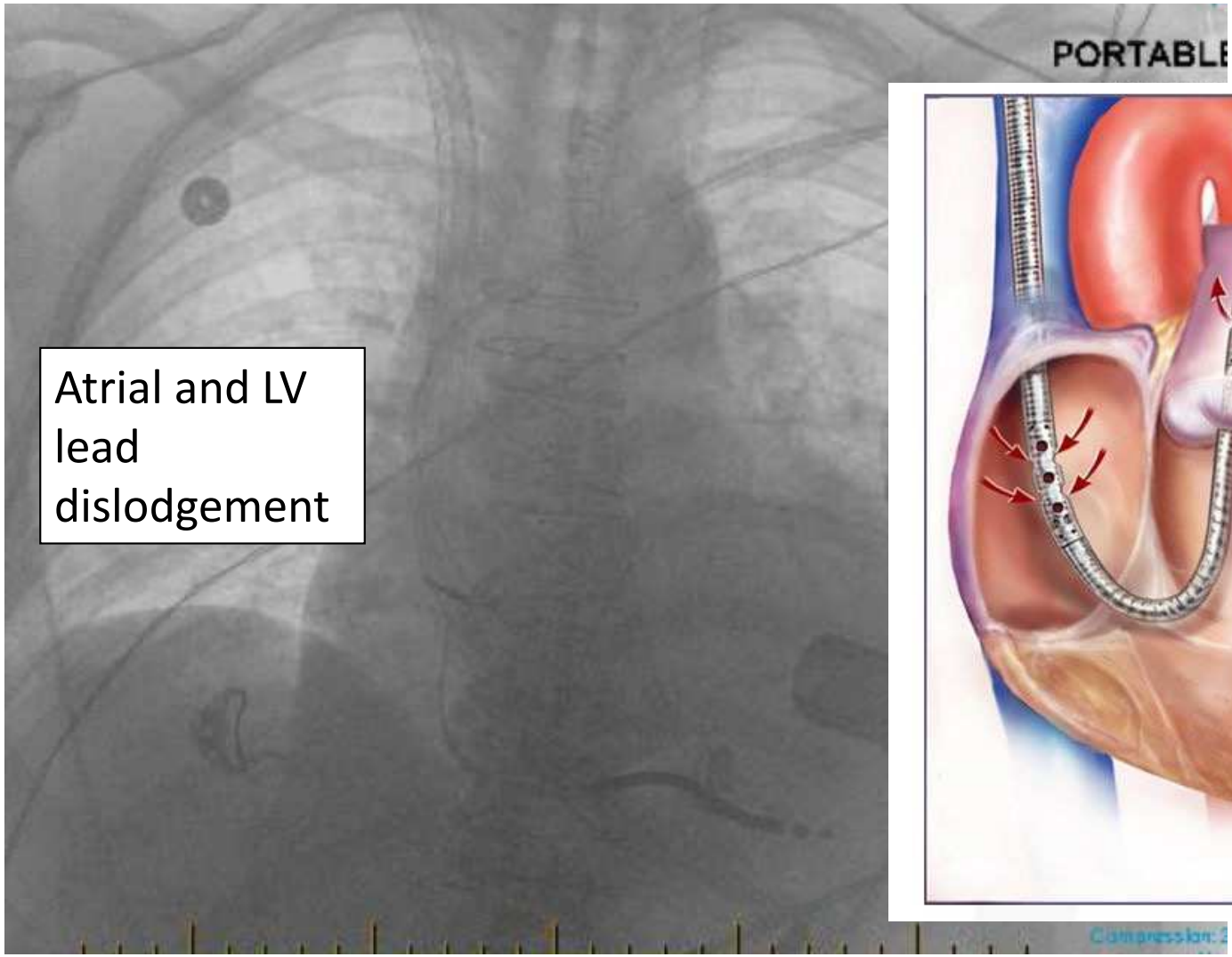
On levo, dobutamine, epi, vasopressin and brought to cath lab in cardiogenic shock with lactate 7 ph 7.1 and flow from LVAD 1.4L/min.

Severe RV dysfunction on TEE



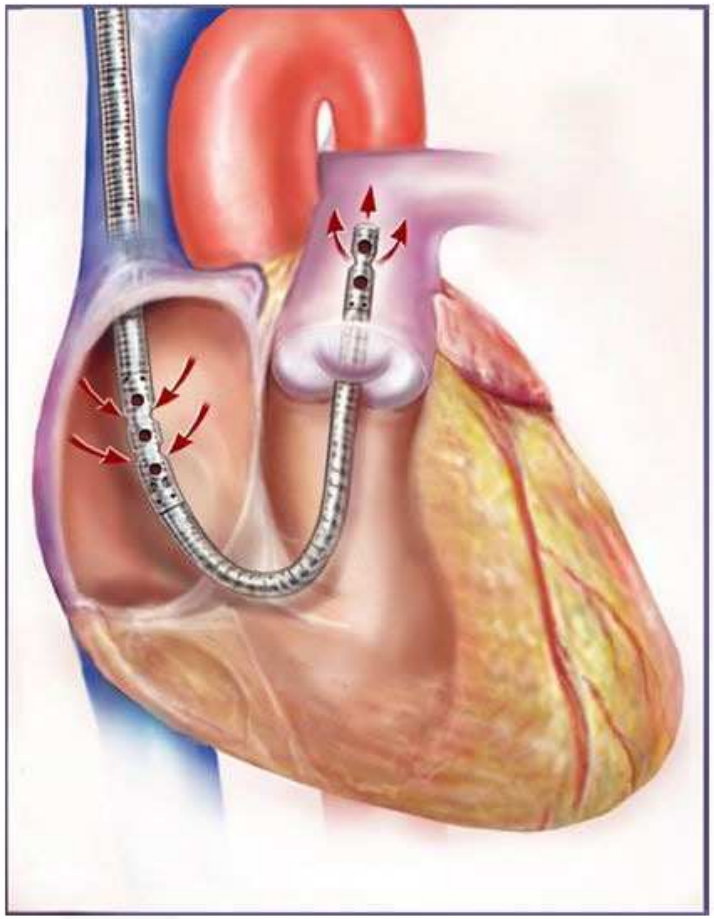

A Axial-Flow Pump
Aorta
Left ventricle
External battery pack
Outflow graft
Skin entry site
System controller
Axial-flow LVAS
Percutaneous lead
To aorta
From left ventricle
Motor
Pump housing
Outlet stator and diffuser
Percutaneous lead
Rotor
Blood flow
Inlet stator and blood-flow straightener
B Fully Magnetically Levitated Centrifugal-Flow Pump
Aorta
Left ventricle
Outflow graft
External battery pack
Skin entry site
Centrifugal-flow LVAS
Percutaneous lead
System controller
From left ventricle
Inflow cannula
Motor
Blood flow
Slide lock
Motor
Rotor with internal magnet
To aorta
Pump chamber





Atrial and LV lead dislodgement

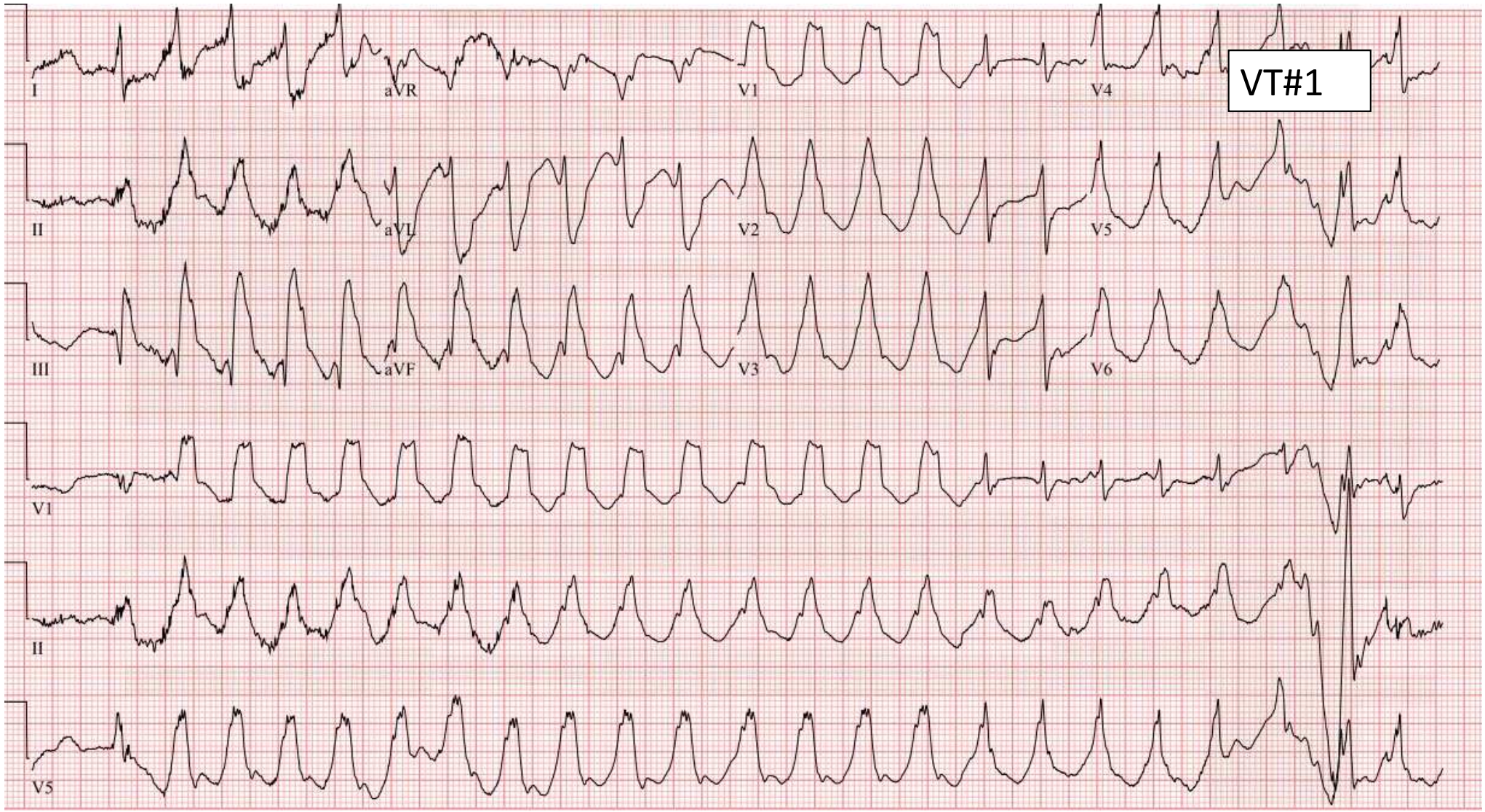
Protek Duo

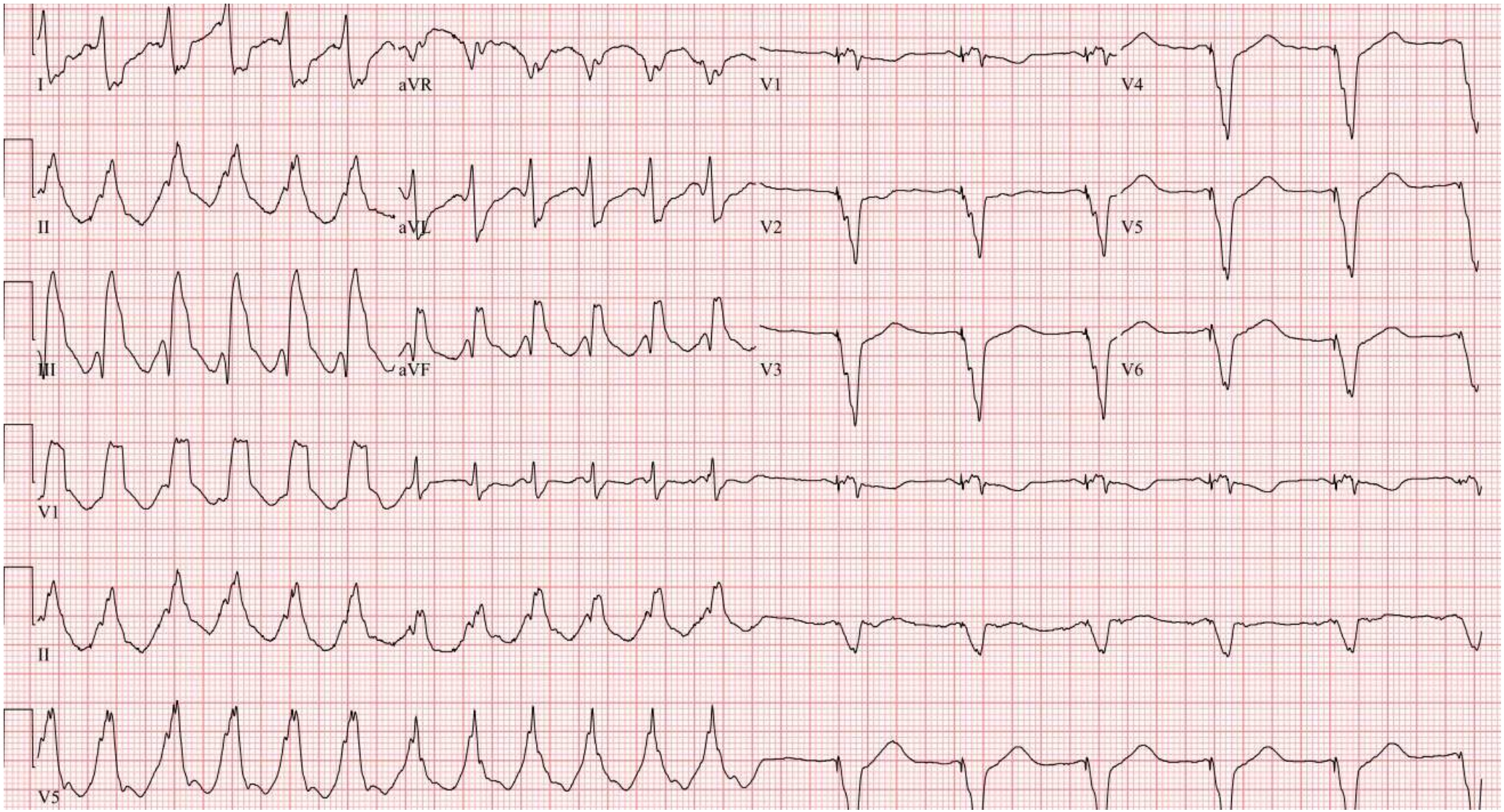


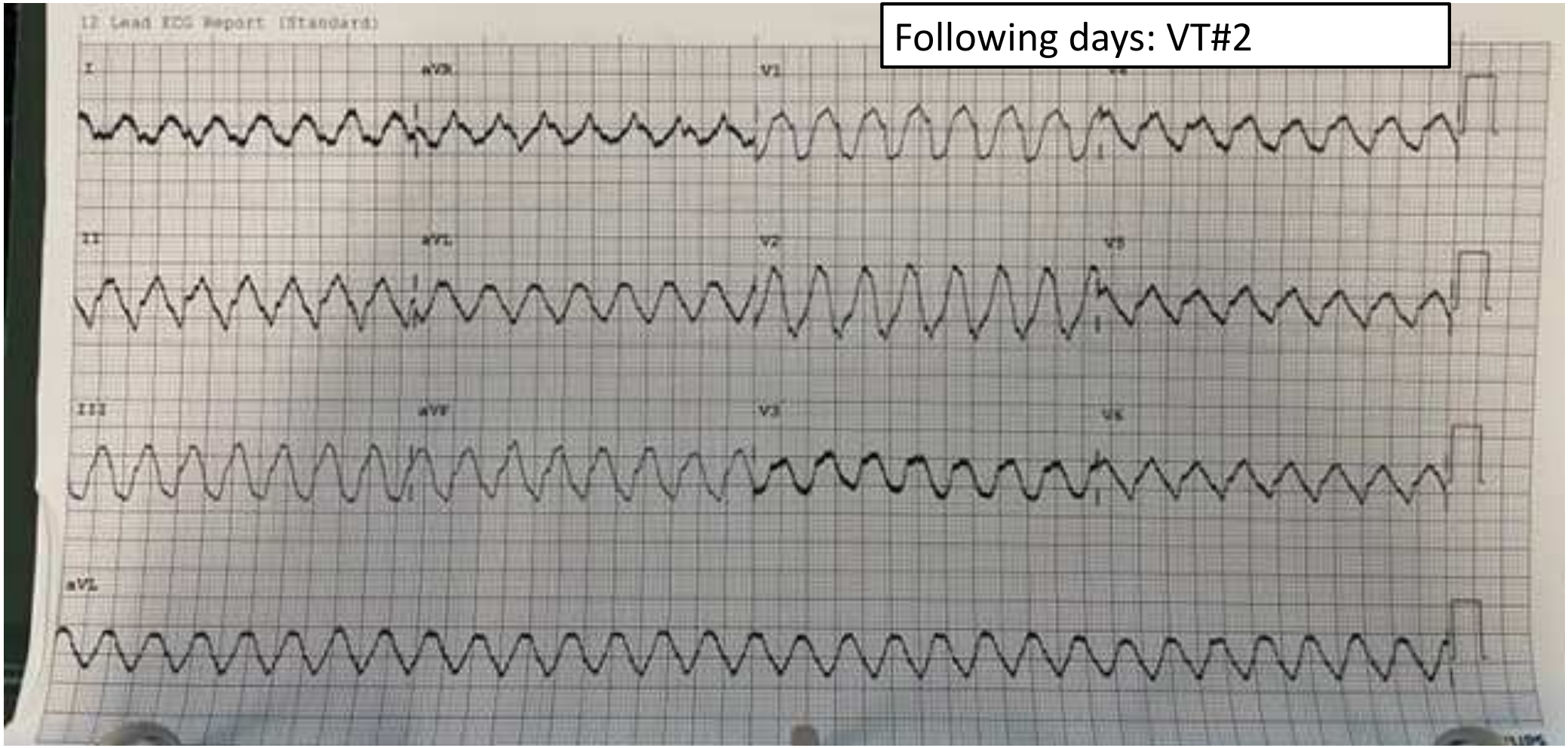
Interrogation in ICU for VT - 8 days post LVAD implant. RVAD was removed yesterday
At my arrival, patient is in monomorphic VT at 220 bpm.
IV amiodarone and lidocaine were started.
After mild sedation, external synchronized cardioversion was successful in terminating the arrhythmia.
On interrogation, the device detected the arrythmia and exhausted all programmed shocks (6).

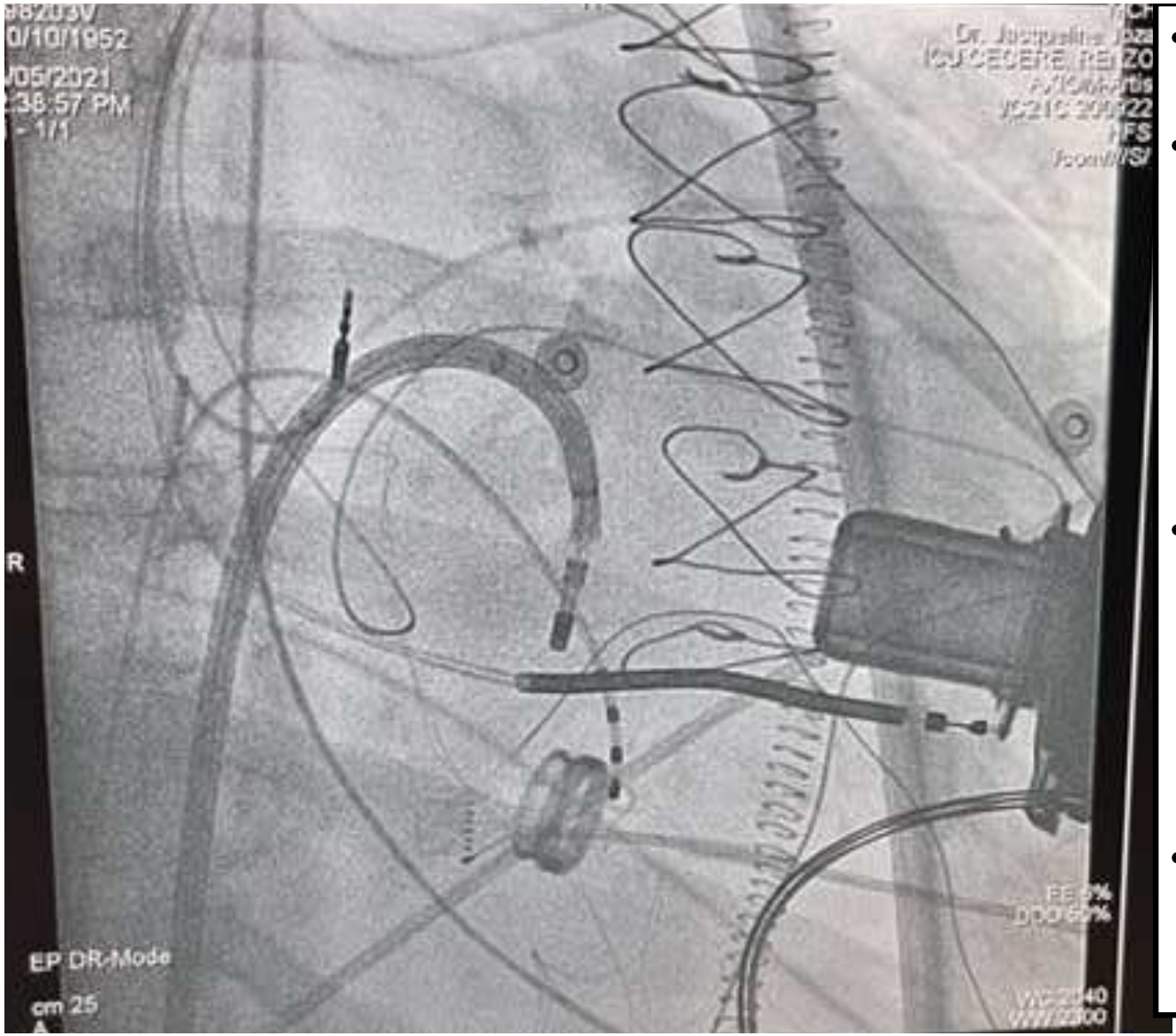
VT recurred shortly after, at different CLs and morphologies. ATP and shocks were delivered by the device.

Plan: - sedation with propofol
- continue IV amio + lido
- Programming left as is with first VT zone at 125 bpm since patient own sinus is running at 97 bpm.

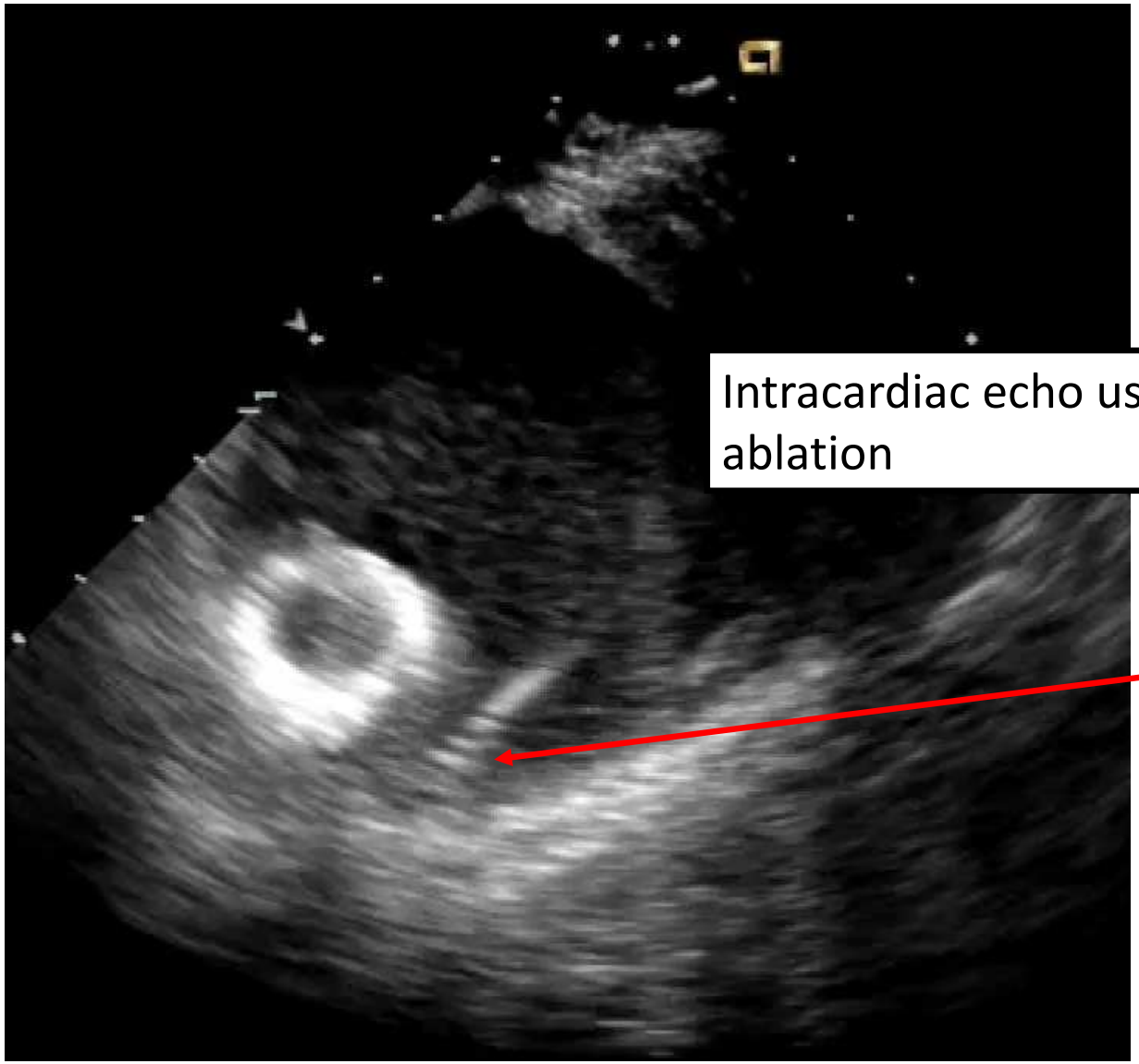

I
aVR
V1
V4
VT#1
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

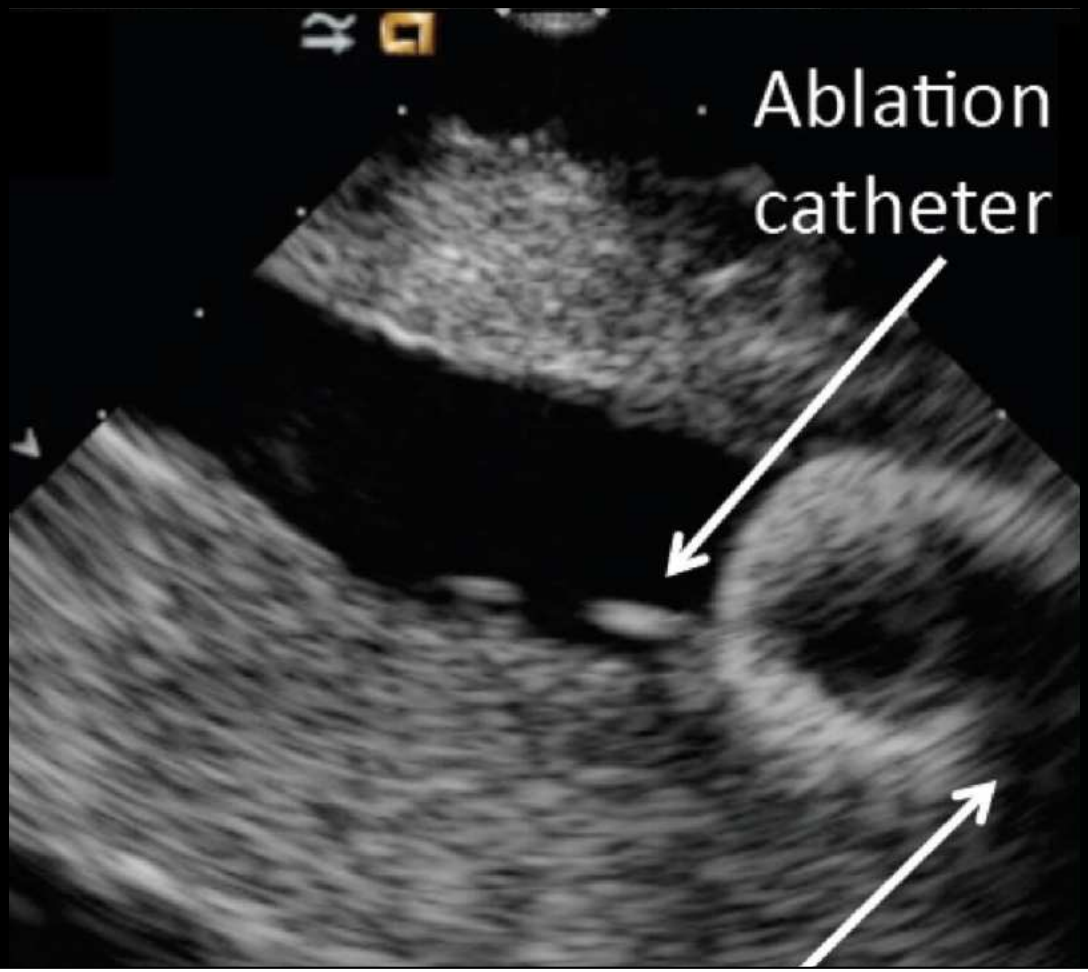

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5


12 Lead ECG Report (Standard)
I
aVR
V1
II
aVL
V2
III
aVF
V3
aVL
Following days: VT#2

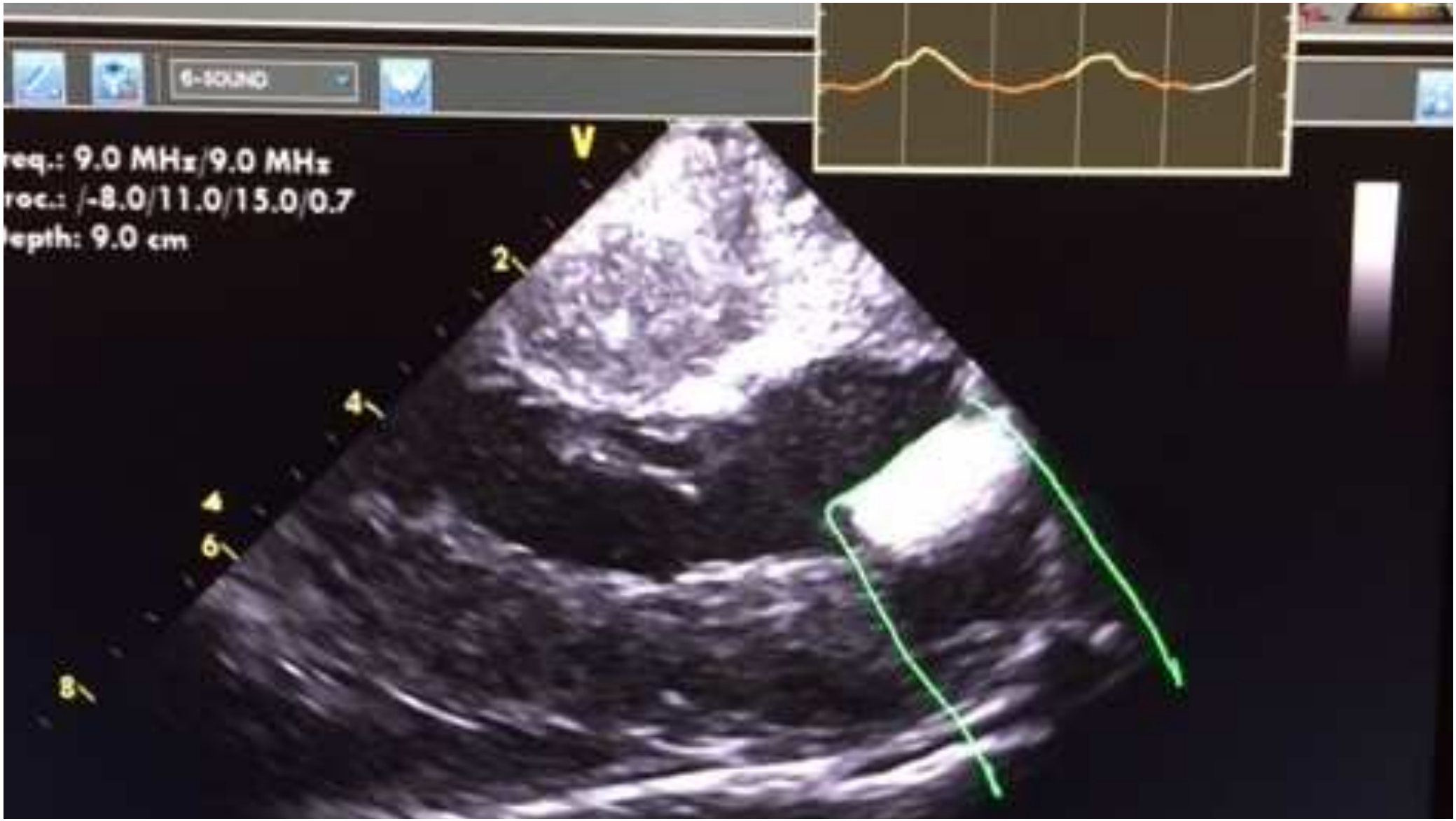


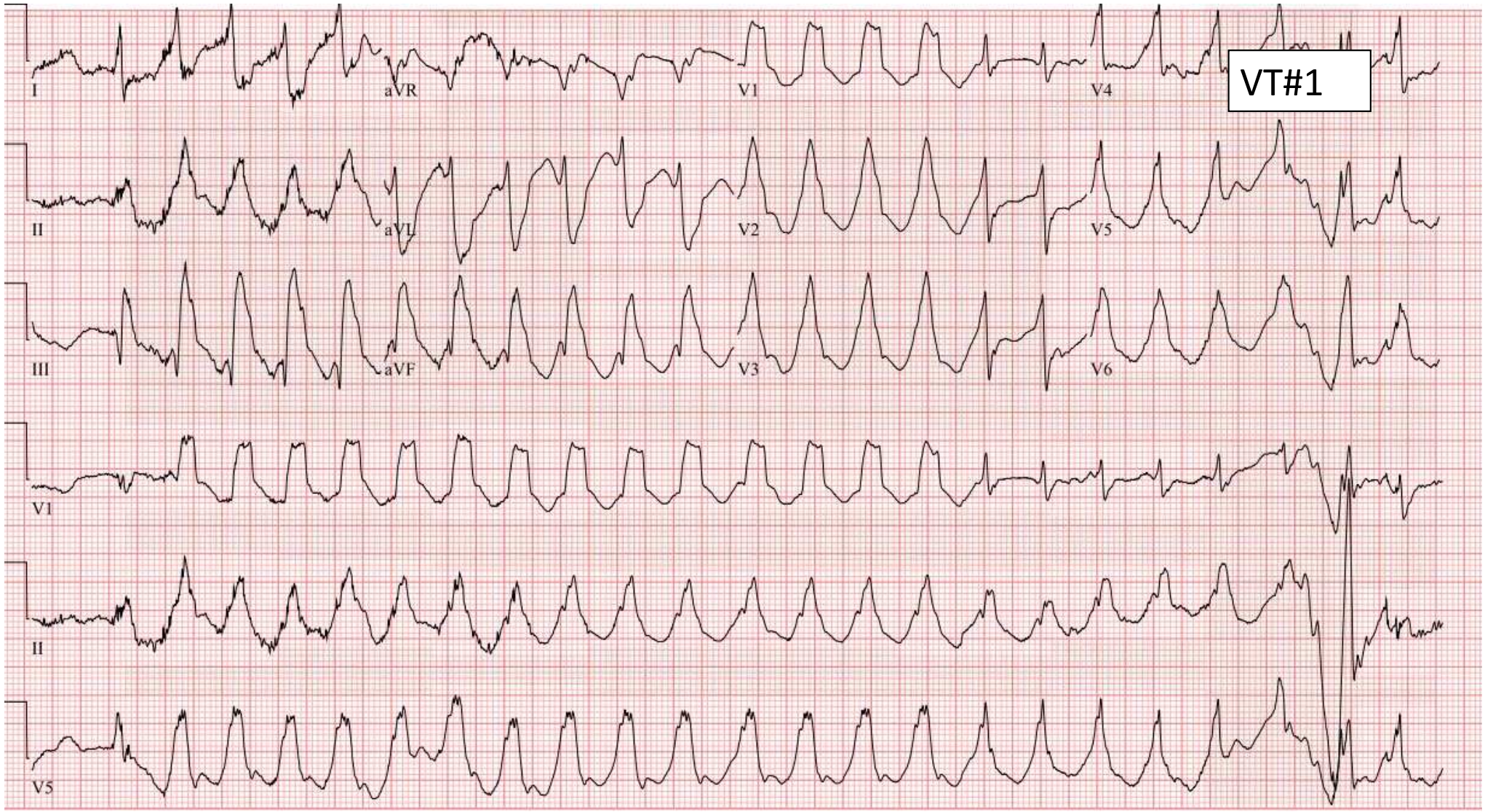
- Transseptal approach preferred
- Thrombus formation at the aortic root can occur due to absence of aortic valve opening (even in pts fully anticoagulated). TEE recommended pre-abl
- Risk of catheter entrapment is low (although we do not advance them beyond the initial portion of the cannula where the turbine is located)
- Invasive arterial monitoring important as absence of pulsatile peripheral pulse

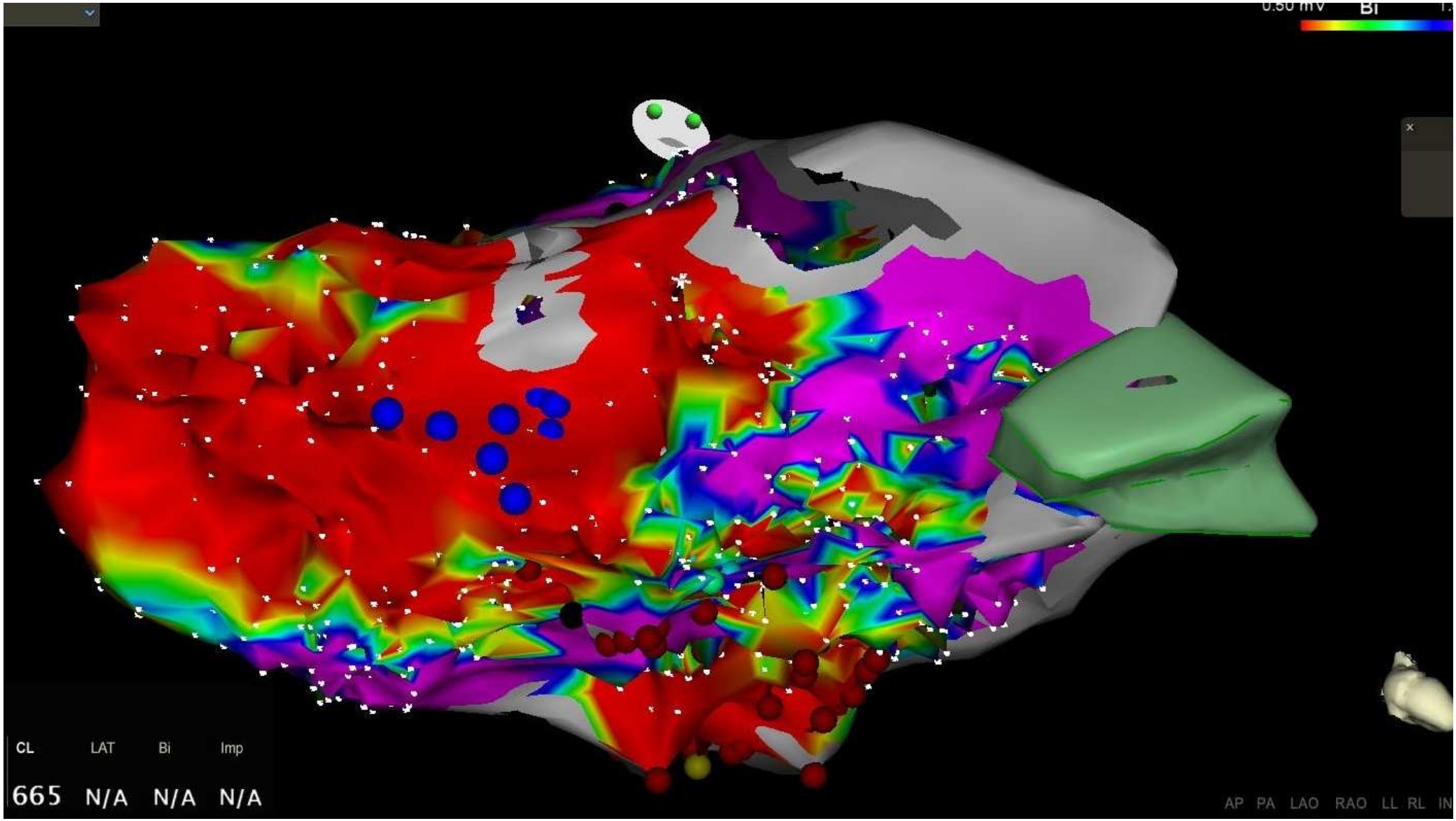

Intracardiac echo used during ablation
Ablation catheter sitting next to inflow

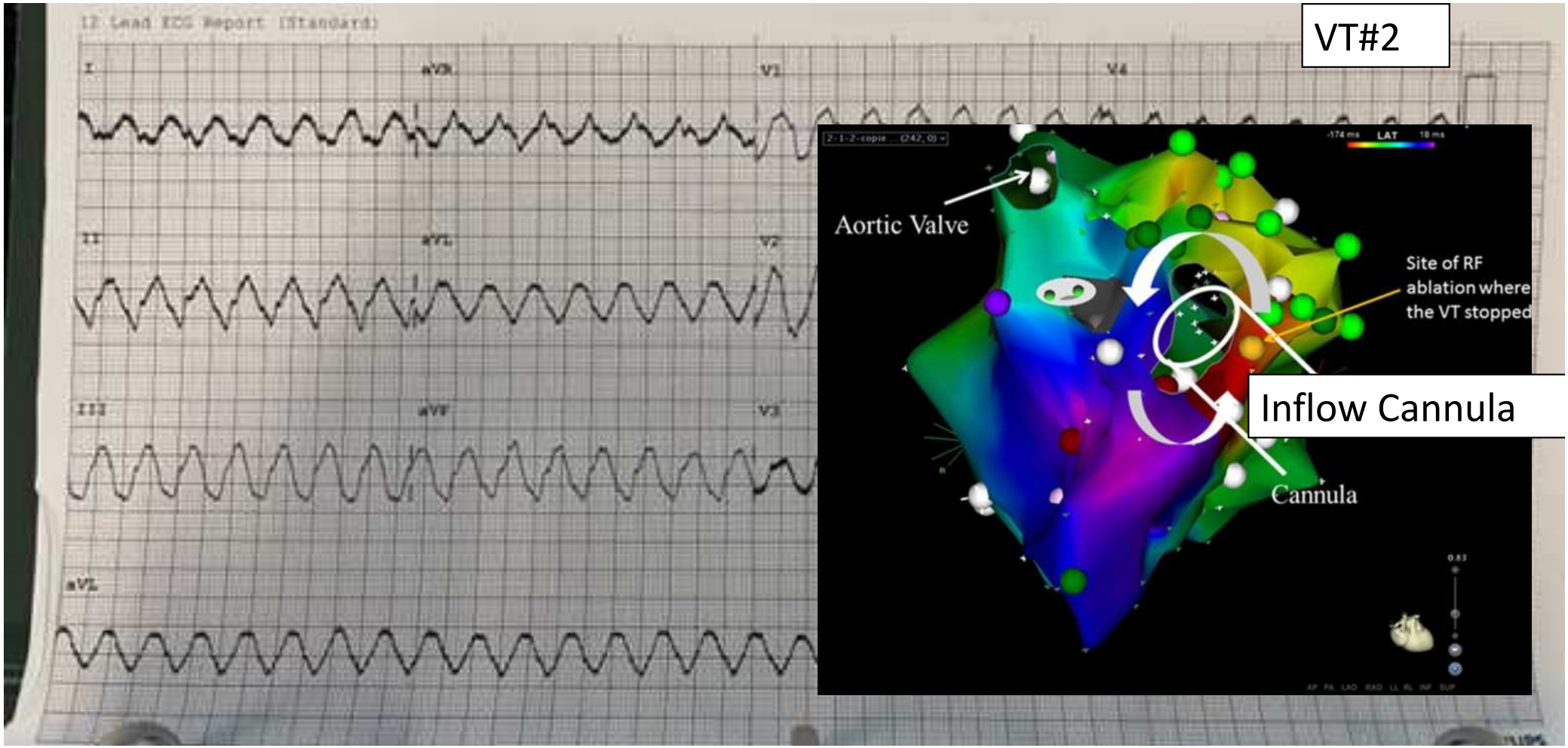


Hypotension and LVAD flow cessation with complete obliteration of the LV cavity. A decrease in the pump speed allowed restoration of the LV size, combined with volume.


eq.: 9.0 MHz/9.0 MHz
oc.: /-8.0/11.0/15.0/0.7
epth: 9.0 cm
V
2
4
4
6
8


I
aVR
V1
V4
VT#1
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5


0.50 mV
Bi
CL LAT Bi Imp
665 N/A N/A N/A
AP PA LAO RAO LL RL IN


12 Lead ECG Report (Standard)
I
aVR
V1
V4
II
aVL
V2
III
aVF
V3
aVL
VT#2
Aortic Valve
Site of RF ablation where the VT stopped
Inflow Cannula
Cannula
LAT

Case 2: 62M - final

Remained in ICU without VA recurrence

Required CVVH from the beginning – eventually transferred for interval dialysis

Went to the dialysis unit for a hemodialysis session: became hypotensive, asystolic, long resuscitation, and eventual anoxic brain injury

Conclusions: VT Storm

1. Ventricular arrhythmias portend a poort prognosis in the HF population

2. Remember the benefits of IV lidocaine as first-line therapy AAD for polymorphic VT/VF and post-myocardial infarction VT, with use in monomorphic VT particularly if proceeding to ablation

3. Consider pre-LVAD treatment of ventricular arrhythmia substrate